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TENSION PNEUMOMEDIASTINUM ASSOCIATED WITH OPERATIONS ON THE NECK

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In 1884 Champneys reported 82 cases of pneumomediastinum in association with tracheotomy operations. Since that time there have been reports from time to time on this condition, but it is one of comparative rarity and its incidence is apt to be forgotten by both surgeons and anaesthetists. Prompt action, when it arises, can be a life-saving measure.

Matthew Spence[1] reports a fatal case associated with positive-pressure anaesthesia in which pressure of over 20 mm. Hg was momentarily applied inadvertently. The mechanism in this type of case, however, is not the same as that associated with neck operations, although bilateral tension-pneumothorax may be the result in either case.

Bowden and Schweizer[2] in their admirable article discuss both types at length.

Anatomy

Some knowledge of the anatomy of the cervical fascia is essential to the understanding of the mechanism of the condition. Of the 3 layers of this fascia only the middle and the deep layers are involved.

Middle Layer (*Pretracheal Fascia*). This forms the carotid sheath laterally and extends to form the fascia covering the scalene muscles and merging with the fascia covering the levator scapulae and trapezius muscles. Inferiorly it extends into the mediastinum to blend with the fibrous pericardium. Thus a puncture in this fascia anywhere in the neck will provide a free passage into the mediastinum.

Deep Layer (*Prevertebral Fascia*). This fuses with the scalene fascia, ending laterally by fusing with the fascia covering the trapezius, levator scapulae and erector spinae muscles. It forms Sibson's fascia over the apex of the pleura. Inferiorly it is continuous with the endothoracic fascia in the superior mediastinum.

Between the middle and deep layers of the cervical fascia in the mid-line is the space containing the trachea and oesophagus, which is an open passage to the superior mediastinum.

Mechanism. A wound in the pretracheal fascia frequently results in a 'uni-directional valve', air being sucked into the mediastinum during inspiration and being unable to escape during expiration. This phenomenon is particularly likely to occur if there is partial respiratory obstruction such as that caused by the kinking of an endotracheal tube. The increase in the mediastinal negative pressure results in a rapid increase in the volume of air being sucked into the mediastinum. As the pressure of the pneumomediastinum increases, air may track along the various tissue planes resulting in emphysema of the neck, face, axilla or thoracic wall. Bowden and Schweizer[2] report a case in which emphysema of the abdominal wall also resulted.

Unilateral and bilateral tension-pneumothorax may also eventuate from rupture of the mediastinal pleura. This appears to occur with greater facility in young children as their tissues are less able to withstand the increased pressure. Thus serious embarrassment of the action of the lungs results, the hypoxia and hypercapnoea rapidly becoming a matter of extreme urgency.

Diagnosis. If the condition is remembered, it should not present untoward difficulty. The first sign is usually laboured abdominal breathing, to all intents and purposes resembling the respiration of acute respiratory obstruction. There will be a discrepancy between the respiratory effort and the movements of the rebreathing

bag. Cyanosis may supervene, with signs of carbon-dioxide excess. If not treated energetically at this stage circulatory failure may develop. There are possibly two reasons for this. Firstly the increased mediastinal pressure may interfere with the venous return and, secondly, the marked degree of hypoxia and hypercapnoea undoubtedly brings about circulatory failure if allowed to persist for any length of time. The breath sounds are lessened and, if X-ray diagnosis is available, this will clinch the diagnosis. If, however, there should be any doubt, needling of the pleura in the mid-axillary line is not a procedure fraught with great danger, and may be a life-saving one.

Treatment. The treatment of choice is to puncture the pleura with a large needle and to withdraw as much air as possible by suction. As the pneumothorax is frequently bilateral it is advisable, especially in the absence of prompt radiographic investigation, to puncture both pleurae. Inflation of the lungs with oxygen should rapidly restore oxygenation and eliminate excess of carbon-dioxide. Subsequently the needles should always be attached to under-water drainage and not removed until it is certain that there will be no further lung collapse. The patient should be nursed at first in an oxygen tent.

CASE REPORT

D.G., a healthy female child of 14 months, was operated upon for the removal of a cystic hygroma of the neck. Premedication—atropine, gr. 1/150. Induction of anaesthetic with nitrous oxide, oxygen and ether; intubated through the mouth; maintenance with nitrous oxide, oxygen and minimal ether, with an Ayre's T-tube.

Operation continued uneventfully for 1½ hours, when partial respiratory obstruction developed due to interference with the catheter mount and the T-tube by the assistant's elbow. This was

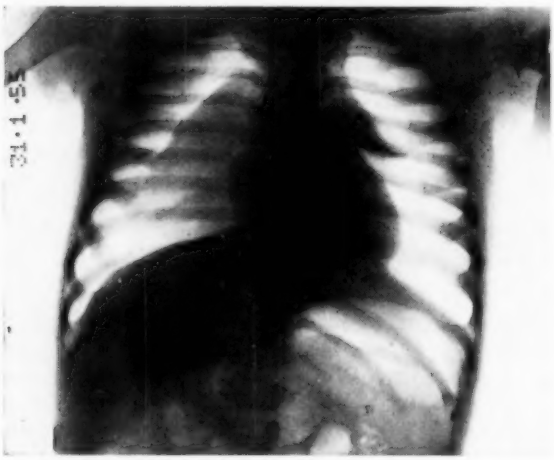

Fig. 1. X-ray showing considerable bilateral pneumothorax. Note bulging of right auricle due to interference with pulmonary circulation.

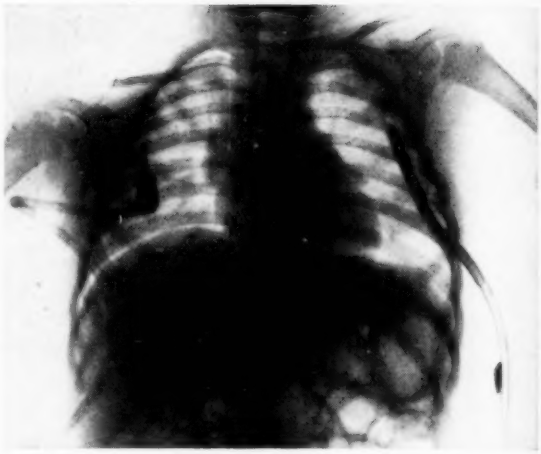

Fig. 2. Condition largely alleviated by bilateral under-water drainage of both pleural cavities.

rectified and the patient appeared to be in a satisfactory condition.

Fifteen minutes later further signs of what appeared to be partial respiratory obstruction developed, with some cyanosis and laboured abdominal breathing. On examination of the tubes no signs of obstruction or kinking could be found. The signs, as was seen a little later, were due to a bilateral tension-pneumothorax. Slight pressure was applied to the rebreathing bag on inspiration as the surgeon was completing the skin sutures. This possibly aggravated the condition instead of relieving it[2]. The endotracheal tube was removed and the trachea well sucked out but this did not improve the patient's condition in any way. The child was cyanotic unless pure oxygen was administered.

On examination of the chest the breath sounds were faint. An X-ray taken within 10 minutes showed a bilateral pneumothorax with the lungs pushed up against the mediastinum. Although the X-ray plate was developed in 5 minutes more, the heart failed at the time that the plate arrived. The circulation was restored within 5 minutes; an incision was made in the 4th interspace on the left and the heart was grasped and squeezed about 60 times per minute[3]. The right pleura was punctured with a large needle attached to an under-water drain and the left chest closed, leaving an intercostal tube attached to an under-water drain. (Figs. 1 and 2). Spontaneous respiration commenced within a short time, but the child did not at any time become fully aware of her surroundings although she moved about and attempted to suck. She was nursed in an oxygen tent and the drains removed on the second day as respiration was satisfactory. Feeding was carried out through a stomach tube.

The patient continued to go downhill and died on the 5th day from irreversible cerebral damage that had been caused by the anoxia.

SUMMARY

The mechanism of tension-pneumomediastinum is discussed in association with operations on the neck. Its complications, especially that oftension-pneumothorax, are mentioned. Emphasis is on prompt and adequate treatment.

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EDITORIAL

PAIN

Pain is a very common manifestation of injury and disease and for the physician it is a useful symptom, since without it many a diagnosis would be difficult or delayed. Much information is available on the subject, though little of the knowledge has passed into general circulation, and there is still much scope for research into the many problems which the subject presents. The problems of pain are well set out, especially from the point of view of the practising doctor, in one of the 'Practitioner' handbooks.[1]

Pain is produced by stimulation of free nerve-endings of fine terminal branches of nerve fibres. By means of stimuli applied to various parts of the body it has been shown that there are two waves of pain, with an interval between them corresponding to the difference in the rate of conduction in myelinated and non-myelinated afferent fibres. However, stimuli applied to the surface of the body are likely to stimulate the endings of many different fibres; thus, however localized the stimulus, the message which reaches the central nervous system will produce sensation representing the summation of effects from many different fibres. The precise mechanism of stimulation of the pain nerve-endings in the skin is not known with certainty, although some change in the permeability of the surface membrane of these nerve-endings appears to be essentially involved.

The effects of pain on the higher levels of the central nervous system are hardly confined to precise channels but are diffuse and generalized. There are no regions of the cerebral cortex specially concerned with reception of pain. The effect of pain is mainly on the central regions which regulate cortical activity; the pain signals play little part in building up cortical or mental patterns, although the cortex does apparently contribute to the perception of pain as a mental event. The ventrolateral nucleus of the thalamus is almost certainly the main perceptive centre for the affective aspects of pain. Lesions of the cerebral cortex do not abolish perception of pain permanently nor do they reduce materially the sensibility to pain-producing stimuli; on the other hand destruction of the lateral part of the thalamus produces analgesia of the opposite half of the body.

VAN DIE REDAKSIE

PYN

Pyn is 'n baie algemene manifestasie van besering en siekte en vir die geneesheer is dit van belang want daarsonder sou menige diagnose moeilik wees of vertraag word. Heelwat inligting is oor die onderwerp beskikbaar, maar dit is nie algemeen bekend nie. Daar is ook heelwat geleentheid vir navorsing oor die menigte vraagstukke wat die onderwerp oplewer. In een van die ‚Practitioner'-handboeke[1] is daar 'n deeglike uiteensetting van die probleem veral soos dit die praktiserende geneesheer raak.

Pyn word veroorsaak deur prikkeling van die vrye senu-ente van die fyn eindvertakkings van die senuweedrade. Deur prikkelings op verskillende dele van die liggaam toe te dien, is bewys dat daar twee pyngolwe is met 'n tussenpose wat gelykstaan aan die verskil in spoed van geleiding tussen toevoersenuweedrade met murgskedes en dié sonder murgskedes. Prikkelings wat egter op die oppervlakte van die liggaam toegedien word stimuleer moontlik die ente van 'n aantal verskillende senuweedrade. Dus, hoe gelokaliseer die prikkeling ookal mag wees, sal die boodskap wat die sentrale senuweestelsel bereik 'n gewaarwording skep wat die totale uitwerking van verskillende drade verteenwoordig. Die juiste meganisme waardeur die pyn-senu-ente in die vel geprikkel word, is nie met sekerheid bekend nie, alhoewel dit waarskynlik is dat een of ander verandering in die deurdringbaarheid van die oppervlakkige vlies van hierdie senu-ente hoofsaaklik daarby betrokke is.

Die uitwerking van pyn op die hoër sentrums van die sentrale senuweestelsel is nie juis tot presiese kanale beperk nie maar is versprei en algemeen. Geen deel van die breinskors is spesiaal by die opneem van pyn betrokke nie. Pyn affekteer veral die sentrale dele wat die skorsaktiwiteit beheer. Die pynprikkels speel nie 'n groot rol in die vorming van skors- of begripspatrone nie, alhoewel die skors waarskynlik bydra tot die gewaarwording van pyn deur die verstand. Dit is amper seker dat die ventrolaterale kern van die talamus die hoofwaarnemingsentrum vir die gevoelsaspekte van pyn is. Letsels van die breinskors neem nie die persepsie van pyn permanent weg nie, ook verminder hulle nie wesenlik die gevoeligheid vir pynverwekkende prikkels nie. Vernietiging van die laterale deel van die talamus veroorsaak egter gevoelloosheid van die teenoorgestelde helfte van die liggaam.

Soos hierbo aangedui stimuleer pynverwekkende prikkels ook ander soorte gewaarwordings, soos bv. die

As indicated above, pain-producing stimuli excite other forms of sensation, such as touch or pressure or heat, and these sensations produced at the same time as pain are probably responsible for accurate localization and discrimination. In the rare condition known as the thalamic syndrome, caused by softening in the lateral aspect of the thalamus, intense pain occurs in the opposite side of the body. Otherwise the central nervous system is insensitive to injury and no pain arises unless sensory roots, or parts of the meninges, or possibly larger vessels, are also involved.

The relief of pain has always been one of the chief tasks of doctors, and it is a well-established principle that if possible it should not be allowed to interfere with the full investigation and treatment of the condition which is causing the pain. The possibility of pain being psychogenic in origin should always be borne in mind. Pain of apparently non-organic origin may be referred to any part of the body. Careful examination of the history, with appreciation of the behaviour of the patient, may enable a conclusion to be reached and a positive diagnosis to be made. The services of a psychiatrist may be essential when no satisfactory organic aetiology can be found, when pains recur at regular intervals, and when ordinary analgesic measures do not bring relief. The pain of functional disease requires treatment just as obviously as pain due to organic disease; and in the correction of the psychogenic disorder not only medicinal treatment may be of value, but also social and moral help.

1. *Pain and its Problems* (1950): The Practitioner, London: Eyre and Spottiswoode.

tas-, druk-, of hittegevoel, en hierdie gewaarwordings wat tegelykertyd met die pyn ontstaan, is waarskynlik verantwoordelik vir presiese lokalisering en die vermoë om te onderskei. In die seldsame kondisie, bekend as die talamussindroom, wat veroorsaak word deur verweking in die laterale aspek van die talamus, kom daar geweldige pyn in die teenoorgestelde kant van die liggaam voor. Anders is die sentrale senuweestelsel gevoelloos vir besering en geen pyn word gevoel nie tensy sensoriese wortels, of dele van die breinvliese, of moontlik groter bloedvate betrokke is.

Verligting van pyn is nog altyd een van die geneesheer se grootste take. Dit is egter 'n erkende beginsel dat, indien moontlik, die verligting van pyn nie moet verhinder nie dat die toestand wat die pyn veroorsaak, volledig ondersoek en behandel word. Die moontlikheid moet altyd in gedagte gehou word dat pyn psigogeen in oorsprong kan wees. Pyn wat oënskynlik nie-organies van oorsprong is, kan na enige deel van die liggaam uitgestraal word. Noukeurige ondersoek van die geskiedenis, met inagneming van die pasiënt se gedrag, kan mens in staat stel om tot 'n gevolgtrekking te kom en om 'n positiewe diagnose te maak. Die hulp van 'n psigiater is waarskynlik nodig wanneer geen bevredigende organiese oorsaak gevind kan word nie, wanneer pyne met gereelde tussenposes terugkeer, en wanneer die gewone pynstillende middels nie verligting bring nie. Die pyn van funksionele siektes het behandeling net so nodig soos die pyn wat aan organiese siektes te wyte is; vir die verhelping van 'n psigogeniese kwaal is nie net geneesmiddels van belang nie, maar ook sosiale en morele hulp.

1. *Pain and its Problems* (1950): The Practitioner, Londen: Eyre en Spottiswoode.

CLUBBING OF THE FINGERS AND HYPERTROPHIC PULMONARY OSTEO-ARTHROPATHY

Nothing in medicine is more puzzling than the clubbed finger. How can one hope to explain it when it can apparently occur in such a variety of conditions as cyanotic heart disease, bacterial endocarditis, a tumour in the lung parenchyma no larger than a marble, acute hepatitis within 15 days, aneurysm of the aorta, intrathoracic goitre, myxoedema, and idiopathic steatorrhoea? Hippocrates unfortunately laid stress on the non-essential changes and today clubbing is frequently incorrectly diagnosed. The basic clinical feature, so well described by Lovibond,[1] consists of an increase in soft tissue beneath and around the bed of the nail, without which clubbing cannot be considered present.

None of the various theories that have been proposed to explain the production of clubbing is satisfactory for all cases, though a few more definite clues are now available. There is an anatomical change in the capillary loops in the nail bed, which are wide and tortuous, while the blood flow through the clubbed fingers is increased (this does not apply to the congenital cases). If the causal intrathoracic lesion can be eradicated the clubbing and heightened blood flow may disappear *pari passu*. On the other hand an increase in blood flow can hardly be the primary factor, since clubbing does not occur in hyperthyroidism, and it would be strange to think of hypothyroidism initiating a rapid circulation.

Hypertrophic osteo-arthropathy is equally obscure. Here there is pain and stiffness in the joints of the limbs, together with pain, swelling, tenderness and even redness of the limbs themselves, especially of the forearms towards the wrists. The underlying intrathoracic lesion may be silent and rheumatoid arthritis diagnosed. Clubbed fingers, however, are always present—in fact we usually consider osteo-arthropathy as a sort of extension of clubbing. Yet the two are highly dissimilar—clubbing is painless (with rare exceptions), osteo-arthropathy usually painful; in clubbing the new tissue is non-osseous and obvious on inspection, in osteo-arthropathy it is periosteal and bony, and needs an X-ray for its diagnosis.

With the increasing incidence and awareness of bronchial carcinoma it would appear that this may now be regarded as the chief cause of osteo-arthropathy, and Semple and McCluskie[2] have recently described 24 examples of such a combination. They call attention particularly to those cases in which the joint symptoms may be the presenting features of a lung cancer. The moral, presumably, is to X-ray the chest in all cases of

slightly atypical 'rheumatoid arthritis' or 'rheumatics in the forearms' occurring in middle age.

Both clubbed fingers and osteo-arthropathy may also be congenital and hereditary, being passed on from parent to child as Mendelian dominant characteristics, in which case, of course, there is no concomitant internal disease. Often the hereditary and occasionally the acquired variety of osteo-arthropathy is combined with an overgrowth of the skin of the face (as well as the hands) producing deep furrowing (cutis gyrata). The combined syndrome has been euphoniously entitled 'pachydermoperiostosis'. Fried,[3] for this and other rather inadequate reasons, has resurrected the confusion of osteo-arthropathy with acromegaly which existed in the mind of Pierre Marie.

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PEPTIC ULCER

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Durban

In 1936 Selye[1] in describing the findings in animals submitted to physical and toxic stress stated *inter alia:* 'Gastro-intestinal ulcers and other manifestations of shock were actually more severe in adrenalectomised than in intact animals and could be lessened by treatment with cortical extracts. These lesions are not mediated through the adrenal; in fact they are actually combated by an adequate adrenacortical response to stressor agents.'

To those of us who have come to accept peptic ulcer as a stress-induced pathological entity it surely must become a matter of some concern that even after possessing such knowledge for close on 20 years our preventive and therapeutic approach should bear so little relationship to our adopted concept of causation. There must be a reason for this; it may be that we have become too tied down by prejudice to contemplate that past therapy, dietary and medicinal, has been based on erroneous theory and that our ideas urgently need reorientation.

In this field an admirable summary of modern therapeutic measures has been compiled by Berk.[2] In his review he features the vagaries, the limitations and the benefits associated with the exploitation of banthine, methonium salts, sodium carboxymethylcellulose, cortisone, ACTH urogastrone, enterogastrone, protein hydrolysates, anion-exchange resins, gastric mucin, and atropine. Having done so he concludes: 'It is apparent that a specific cure for peptic ulcer is still not at hand.' It is of extreme interest that in this comprehensive review reference to antacid therapy is conspicious by its absence. What the account does reveal is an earnest attempt to give full value to the stress factor and to evolve a therapeutic approach which might adjust the stress as well as the physical and bio-chemical disharmony engendered by such stress or by failure of systemic anti-stress defence.

THE REACTION TO STRESS IN RELATION TO PEPTIC ULCER

In the past we have been led to devote too little attention to the patient as a whole and too much to his ulcer. This being so, it behoves us in the first instance to crystallize in our minds the picture of what constitutes normal reaction to stress, and having done so we are then much better equipped to assess and rectify systemic disharmony indicative of failure of defence against stress.

Normal reaction to stress should be too well known to require lengthy elaboration in such a discussion as this. Cannon[3] has made it common knowledge that adequate reaction by the human economy is represented by an outpouring of adrenaline and a mobilization of sugar into the blood stream. These reactions represent cause and effect, but before the latter eventuates there are numerous hormonal and metabolic activities to be invoked, all of them contributing to the final physiological result and any one, in failure, conditioning the emergence of pathology such as is represented by peptic ulcer.

All digestive stimuli of whatever nature, be they physical or psychological, motor or sensory, visual, olfactory or gustatory, eventually reach the cerebral cortex, whence responses are relayed to the thalamus and hypothalamus. From the latter, impulses are transmitted along the vagus and sympathetic nerves, the latter catering for the outpouring of adrenaline.

In addition to the neurogenic stimuli arising in the autonomic centres in the hypothalamus Hume[4] has adduced evidence that a hypothalamic hormone is formed and that this exerts a stimulatory effect on the anterior pituitary, which in turn by virtue of secreted ACTH sets the suprarenal cortex into active production of its own particular hormones. Hume has also adduced some evidence suggesting that an injection of adrenaline acts on the anterior pituitary in a manner indistinguishable from that of the hypothalamic hormone. If this be so it becomes a matter of interest that a reaction starting in the suprarenal medulla should induce defence response through devious channels at the ultimate level of the suprarenal cortex.

The above findings have been confirmed by Selye[5] and by Ungar,[6, 7] and all investigators are agreed that the ultimate goal to be reached is the prevention of cell-breakdown and of histamine release. In the *British Medical Journal* of 5 March 1947 a sub-leader appraising

research carried out by Ungar in connection with toxic and mechanical injury, summarized as follows:

'Several stages of the argument still need confirmation but it seems probable that normal response to injury is somewhat as follows. The anterior pituitary in response to the stimulus of injury increases its output of ACTH; the adrenal cortex responds with increased activity; the spleen, stimulated by the suprarenal cortical hormones, increases its output of splenin; splenin activates anti-trypsin, which is present in normal serum but which is comparatively inert until combined with splenin; activated anti-trypsin decreases breakdown of protein and thus frustrates the release of histamine; and finally the reduction of circulating vasodilators such as histamine decreases the bleeding time and capillary fragility and permeability which are such constant features of histaminic intoxication.

'It seems not improbable that a new window has been opened through which may be viewed from a fresh angle many problems both of surgery and of general medicine'.

Ungar's research did not stop here. He devised a method of standardized trauma which, when applied to the full, was invariably lethal to the research animal. He was able to show that the lethal effects of such standardized trauma could be neutralized in 95% of cases by giving the animals an injection (*a*) of serum from an animal which had been submitted to a sub-lethal degree of trauma or (*b*) an injection of vitamin C of not less than 100 mg./kg. given within 15 minutes of the infliction of the trauma. He submitted *all* the animals, both those dying and those surviving, to autopsy and macroscopical examination. In all animals which succumbed there were found numerous gastro-intestinal ulcers and haemorrhages and not infrequently actual perforation. In those which survived as the result of administration of serum or vitamin C ulcers were either absent or of minimal degree.

Reverting to the question of histamine: Best[8] has drawn attention to the close integration of this protein derivative into the process of normal digestion. He reminds us that it is recoverable from gastric contents and emphasizes the close analogy of histamine to gastrin as digestive activators. In fact he finds it difficult to differentiate one from the other in that both are inactivated by histaminase and that either pushed to excess is capable of producing hypersecretion of gastric enzymes and eventual haemorrhages and ulceration.

Histamine excess, as is well known, calls up the adrenaline reserves and if these are not freely available histaminic damage becomes inevitable. This may have an important bearing on haemorrhage. With free production of adrenaline as a defence reaction there develops an increased coagulability of the blood; but if defence fails and adrenaline is supplanted by histamine, blood coagulability diminishes and capillary fragility and permeability increase.

The neurological control of digestion is vested in the autonomic nervous system, the vagus being responsible for the intensification of secretion and motility, whereas the sympathetic exercises restraint over these functions.

In the stomach itself there is evidence of sympathetic aberration as recorded by Alvarez,[9] who states: 'Research by Barclay and Bentley has shown that as in the kidney so in the stomach there is in the submucosa a layer of vessels through which blood can be shunted in such a way as to leave areas of the mucus membrane anaemic. This shunt is opened up under stress, and the mechanism like hypermotility and hypersecretion may, therefore, be intimately associated with psychic trauma and could easily explain the formation of peptic ulcers.'

This becomes all the more understandable when viewed in conjunction with the finding of Babkin[10] that *pari passu* with stress-induced accentuation of vagal effects there is thereby produced not only hypersecretion of acid and pepsin but of histamine as well. Nor are these the sole ill-effects of vagal hyperactivity, and as consideration of this involves the question of mobilization of blood sugar, the whole of this subject as it pertains to stress and to the genesis of peptic ulcers will have to be reviewed in some detail.

Mobilization of glucose must depend to a great extent on availability of supplies, and this in turn is subject to the controlling influence of a number of closely integrated functions. It is not sufficient for the purpose that the patient should take liberal quantities of carbohydrate. Control of the storage and utilization of sugar is dependant on adequate production of insulin. Moreover, in the emergency of stress steroidal hormones undertake the role of gluconeogenesis and glycogen storage, the glucose being derived largely from fat and protein. These hormones moreover are powerful insulin antagonists.

McKee *et al.*[11] have recently shown that vitamin C plays a most important part in this hormonal reaction to stress. They found that in normal guinea-pigs, starved for 24 hours, an injection of 3 c.c. of eschatin (suprarenal cortical hormone) given in divided doses produced over a 7-hour period an increase of glycogen storage in these animals amounting to 28 times that found in untreated controls. In similarly treated scorbutic animals, not only was there no increased deposit of liver glycogen but there was an actual decrease. As mobilization of sugar is a prime necessity in adequate defence to stress, vitamin-C quota begins to loom prominently in our thesis.

Thanks to the researches of Giroud[12] and Sayers[13] it is now common knowledge that steroidal hormones generated in the patient as a reaction to stress or administered orally or parenterally as cortisone or ACTH make great demands on vitamin-C storage, and in this respect depletion is particularly accentuated in the suprarenal cortex. Therefore, as peptic ulcer patients have been presumably drawing on these hormonal reserves persistently and continuously, it becomes a reasonable assumption that these patients may in doing so have reduced their storage of vitamin C to vanishing point. This we have known to be so for many years, a conservative estimate being that up to 6,000 mg. will be required to produce saturation of storage in the average case. It might be more than significant that this figure duplicates the dosage employed by Ungar in protecting his guinea pigs from the lethal effects of trauma. Where, moreover, mobilization of sugar is a priority, adequacy of vitamin C becomes indispensable.

From the foregoing it will be apparent that the build-up of sugar and its mobilization as a defence measure are by no means immune from breakdown, and in few conditions is this such a common finding as in peptic ulcer.

In 1945 Abrahamson[14] drew attention to the fact that

peptic-ulcer patients were in a great many instances victims of hypoglycaemia. His findings have been confirmed by Beck,[15] who found 51% of a series of 47 proved cases of peptic ulcer to be hypoglycaemic. Among 40 others with free hydrochloric acid but no ulcers 12·5% had hypoglycaemia. This author emphasizes that control of hypoglycaemia is an important factor in the management of duodenal ulcer. Pock-Steen,[16] in his discussion of the stress-induced leiodystonia syndrome, makes capital of the findings that hypoglycaemia is a prominent feature and peptic ulceration not uncommon. Recently Portis[17] has recorded that in individuals who are victims of severe or prolonged psychological stress, hypoglycaemia is a frequent and significant finding. He attributed the low blood-sugar to hyperinsulinism secondary to right vagal pancreatic stimulation. He would seem to have proved his thesis by being able to adjust blood-sugar and disordered psychology by administration of atropine. It is true that, so far as we know, these patients were not suffering from peptic ulceration, but the stress background was identical in most respects. Gilman confirms the presence of hypoglycaemia in ulcer patients (personal contribution as yet unpublished).

At first sight it may appear to the reader that the relationship of hyperinsulinism and peptic ulcer is being over-emphasized. Let us therefore view the matter from an entirely different angle. How many of us can truthfully declare that he has treated a patient for peptic ulcer who at the same time was suffering from diabetes mellitus? The writer has no recollection of such a happening, and colleagues of long experience agree that such double pathology is a rarity at least. Abrahamson in his series encountered 3 patients with peptic ulcer who had high blood-sugars. He was, however, a careful investigator and submitted these patients to sugar-tolerance tests. The immediate response to the administration of glucose was truly that of a diabetic. This phase was in all 3 cases rapidly followed by a fall of figures to hypoglycaemic levels, indicating the ability of the patients to produce insulin to excess in response to the stimulus of circulating sugar. They were in effect examples of dysinsulinism; and it is recorded that with diet as for excessive production of insulin and antacids ulcers healed rapidly and perfectly with simultaneous adjustment of the blood-sugar to normal levels.

It should never be forgotten that persistent hypoglycaemia is a stressor factor of considerable magnitude. In the researches of Bulato and Carlson[18] it was evident that it stimulated the vagus to redoubled activity in a vicious circle of some gravity.

In view of the foregoing, and accepting that peptic ulceration is the direct or indirect result of stress, the finding in these cases of hyperinsulinism and hypoglycaemia is strong presumptive evidence of failure of hormonal defence, bearing in mind that these hormones are insulin antagonists and are indispensable for gluconeogenesis and glycogen storage such as are characteristic of a normally-constituted defence-reaction to stress.

Sandweiss *et al.*[19] confirmed in a large series of proved cases of peptic ulcer that there was a constant and significant depreciation of the urinary excretion of these hormones, and furthermore that the figures recorded improved as healing took place until ultimately they approximated closely to the average level determined in an equally large number of normal controls. Some indirect confirmation of this finding is contained in the observation of the writer that most ulcer cases exhibit a greatly heightened degree of capillary fragility. Such fragility has been demonstrated by Robson and Duthie[20] to be pathognomonic of suprarenal cortical failure and, as we know, it is also characteristic of histaminic intoxication. It is gratifying to know that this fragility is readily eliminated by administration of cortical hormones.

Critical analysis of the foregoing must lead to certain definite conclusions. They are on the one hand that the hypothalamus is the head ganglion of the autonomic nervous system, and on the other hand that it is the centre through which is inspired the pituitary-adrenal chain defence-reaction to stress. When all factors are working in unison physiological harmony is achieved but, should the chain reaction break down or autonomic-nerve supremacy take control, pathology emerges. Certain schools of thought believe that peptic ulcer arises as the result of autonomic-nerve overactivity, others that ulcers are produced by excess production of adrenal cortical hormones and their maleficent influence on gastric secretion and motility. This article endeavours to show that disharmony of function and associated metabolic distortion are the villains in the piece.

HORMONE, VITAMIN C AND ATROPINE IN THERAPY

If we are right, then, the prerequisites of successful therapy must comprise steroidal hormones, vitamin C, and vagal suppressants. Frequently, and with apparent justification, we are warned against the exploitation of steroidal hormones in ulcer therapy. Such warnings are based on the belief that the action of the hormones in intensifying gastric motility and secretion is the potent factor in promoting disaster. We believe that the intensification of stress by hypoglycaemia plus the severe depreciation in vitamin-C storage are the most important factors in the genesis and perpetuation of the ulcer state. On this basis, when the composite picture of ulcer pathology is unfolded and full value is given to each of a number of diverse features, such warnings if persisted with become an insult to the intelligence.

These hormones should become first choice in ulcer therapy, always provided that in addition (*a*) the patient's storage of vitamin C is brought up to saturation point, (*b*) that steps are taken to neutralize vagal overactivity such as is conditioning hypermotility, hypersecretion, hyperinsulinism and excess release of histamine, and (*c*) that certain adjustments to the waterelectrolyte balance necessitated by hormonal therapy are duly catered for. As these hormones promote potassium excretion and sodium retention, intake of these salts must be closely scrutinized.

As one views the therapeutic problem choice of hormones must lie between (*a*) ACTH, (*b*) cortisone, (*c*) natural adreno-cortical hormones. With each of these, vitamin C on the generous scale advocated by Ungar becomes a *sine qua non*. Moreover, as the vitamin

acts more potently in an acid medium antacids should be used sparingly.

Vagal overactivity must be brought under control, for which purpose atropine would seem to be the agent of choice, combining as it does the 3 virtues of cheapness, of effectiveness, and of confining its activity strictly to the parasympathetic nerves, thereby leaving the sympathetic unhindered in its function of releasing adrenaline for the purpose of mobilization of sugar and inauguration of the hormonal chain defence-reaction. Reactivation of defence will ensure frustration of histamine release and it is certain that in the composite therapy vitamin C will add prestige to its benefactions by promoting healing.

To recapitulate, it is confidently believed that therapy as outlined above will cater for the following desiderata:

1. There will be provided a more effective distribution of essential hormones.
2. Such hormonal augmentation will promote gluconeogenesis and glycogen storage as a defence reaction to stress.
3. Vitamin C will be utilized as a potent factor in the perfection of liver storage of glycogen.
4. Hormones will reduce the inflammatory reaction at the ulcer site, counteract histamine release, and produce local conditions favourable to the healing activity of vitamin C.
5. Reduction of the inflammatory reaction conditions reduction of oedema at the ulcer site, and so may assist relief of pyloric obstruction and pain.
6. Frustration of histamine release will diminish capillary fragility and permeability, increase coagulability of the blood, and thereby minimize tendency to haemorrhage from the ulcer crater. (The inflammatory reaction of course owes its distinctive characteristics to our old friend histamine.)
7. Reduction of histamine release will cater for reduction of excess secretion of acid.
8. Belladonna in the composite therapy will suppress vagal overactivity and thereby subdue hypermotility in the ulcer field, hypersecretion and hyperinsulinism, with its unpleasantand harmfuls tressor factor, hypoglycaemia.
9. The sympathetic contribution to digestive harmony will we hope remain undisturbed, and it is conceivable that the mucosal shunt and patchy mucosal ischaemia may thereby be abolished.

THERAPEUTIC MEASURES AND RESULTS

In 52 of 54 cases so far treated, therapy has comprised (1) 3 c.c. of adreno-cortical hormones mixed with 1,000 mg. of vitamin C given intravenously daily for 3 weeks and thereafter every other day for a further 3 weeks. (2) At the same time oral therapy consisting of 15 gr. of potassium citrate with 10 mm. of tincture of belladonna has been administered thrice daily for the full 6 weeks.

In 2 cases, both of gastric ulcer, ACTH has been used.

Dietary restrictions have been minimal, even in the presence of haemorrhage, but alcohol and tobacco have been strictly rationed.

No patient has been bed-rested or hospitalized except in emergency or at his own request. The majority have remained ambulant and gainfully employed during treatment, and it may be emphasized without fear of contradiction that the psychological uplift engendered by being enabled to continue as wage earners is impressive to a degree.

Over a period of 5 years only 3 patients, none of whom were prepared to submit loyally to the imposed therapeutic discipline, have returned for further treatment.

The most impressive symptomatic reaction to therapy is the rapid and dramatic disappearance of ulcer pain. Unless we ensure that the dosage of vitamin is generous throughout this may engender a feeling of false security and mask the intrusion of a leak or perforation. We can conscientiously affirm that in none of our cases, and in spite of warnings to the contrary, has any complication of this nature been encountered. In fact, in 4 patients active haemorrhage was present when therapy was begun. In each instance bleeding came rapidly under control without recourse to transfusion of blood or other aids to haemostasis.

In 3 instances only has it become necessary to invoke surgical aid. In one case stenosis associated with a juxta-pyloric ulcer precluded the attainment of a successful physiological result, and in the two others posterior ulcers penetrating into the pancreas refused to heal. (Sandweiss recorded 3 of 4 cases treated with cortisone or ACTH as being failures and therefore referred for operation. All 3 cases were examples of ulcers penetrating into the pancreas. Such ulcers are notoriously resistant to all therapy. One hazards the opinion that pancreatic enzymes set free by the ulcer digest the walls of the unprotected ulcer crater.)

CASE REPORTS

It is now proposed to make brief reference to a few of the cases that are illustrative of some of the more intriguing clinical features.

Details of Case 5 are accompanied by serial X-ray pictures illustrating response to therapy consisting of the forbidden ACTH and vitamin C with adopted adjuvants.

Case 1. *Duodenal Ulcer with Haematemesis*

G.G., aged 43, was the first to submit himself to the therapy. In 1948 he had suffered from severe haemorrhage from a duodenal ulcer, necessitating hospitalization and transfusion of blood. He was then treated by strict dietary regime of Lenhartz type and intensive antacid and antispasmodic therapy. Healing was attained and he resumed his profession as an attorney.

In January 1950 he reported to the consulting rooms as the result of a recurrence of ulcer symptoms culminating in a sharp haemorrhage. He refused even to contemplate surgery, influenced understandably by the fact that his father had succumbed to such an operation. He agreed to undertake the role of guinea-pig, to remain at work, and to forbear from the adoption of any dietary restrictions.

He became symptom-free within 5 days except for heart-burn when he drank tea, and full radiological healing was attained in 7 weeks. He has been most cooperative in taking his follow-up ration of vitamin C, has had no necessity to take belladonna, and needless to say is most appreciative of his continued maintenance of digestive health.

Case 2. *Gastric Ulcer*

Mrs. R., the wife of a colleague, had had ulcer symptoms for a long time. Radiological confirmation of an ulcer in the pyloric antrum was obtained in the spring of 1951 and she was hospitalized

and submitted to an intensive course of dietary, antacid and antispasmodic therapy without any abatement of symptoms or evidence of clinical healing.

As the ulcer was gastric and there was a low but not histamine-fast secretion of acid, this, coupled with the site of the ulcer and the age of the patient, inevitably raised the question of possible malignancy. The ulcer was viewed gastroscopically and pronounced simple and not malignant.

As treatment on old-established lines by her medical attendant had failed to promote healing one's suggestion that hormonal and vitamin therapy should be tried was agreed to. In spite of warnings to the contrary (ill-founded in one's opinion) it was decided to administer ACTH in conjunction with vitamin C, the former in dosage of 7½ mg. intra-muscularly 6-hourly and the latter of 1,000 mg. intravenously daily. Oral belladonna and potassium citrate completed the therapeutic attack.

Most gratifyingly the ulcer healed steadily and at the end of 6 weeks healing was completed. To remove all possible doubt the patient was examined radiologically by two separate experts in Durban and one in Johannesburg. All three agreed that evidence of ulcer was negative. There has been no recurrence of ulcer subjectively or objectively.

It is an interesting commentary on this case that ACTH, pronounced dangerous when administered to victims of peptic ulcer because of the probability of induction of haemorrhage or perforation, promoted healing when combined with adequate dosage of vitamin C.

Case 3. *Juxta-pyloric Ulcer with Pyloric Obstruction (Spasmodic)*

Mr. A., aged 63, presented himself in March 1952 from Swaziland with a history of epigastric pain over a period of months definitely related to the taking of food and latterly accompanied by persistent vomiting. On occasions the food rejected contained substances taken the day previously. He had obviously lost a lot of weight, and being in the age-group for malignancy one was naturally unhappy about him. These apprehensions were not modified on receiving a radiological report of 6-hour total retention of barium in the stomach, and of apparent complete pyloric stenosis designated as 'due to cicatricial contraction, spasm or new growth but impossible to define'.

It was decided to treat him as a case of simple ulcer with pyloric spasm in the hope of getting relief and a more exact diagnosis. Cortical extract, vitamin C, belladonna and potassium citrate were therefore administered with early and sustained relief. X-ray after 6 days of this therapy disclosed complete relief of pyloric spasm reflected in normal gastric emptying, and definition of a juxta-pyloric duodenal ulcer.

The patient refused to stay in hospital after 3 weeks' therapy, by which time he was symptom-free and X-ray revealed well-advanced healing. He returned to Swaziland with full details for his local doctor to continue therapy, but was apparently not too easy to handle. Six months later he returned with a recurrence of ulcer symptoms but no vomiting. Radiological report was to the effect that there was no pyloric stenosis, the previous ulcer had disappeared, and a fresh active ulcer was present in the posterior wall of the first part of the duodenum.

This time he decided to cooperate for the 5 weeks necessitated by therapy, and when seen 8 months later was adhering to follow-up therapy with meticulous care and a symptom-free result.

Case 4. *Duodenal Ulcer with Hypoglycaemia*

G.A., a clerk in the railway service, had had ulcer symptoms for several months, but was much more concerned with the fact that he had bouts of trembling and sweating several times a day for no obvious reason and attributed to hysteria. Barium X-ray showed an active duodenal ulcer, and resting blood-sugar of 60 mg.%, with figures after taking 50 g. of sugar of 155, 80, 65 and 55 mg.% at half-hourly intervals, confirmed the suspicion of hypoglycaemia.

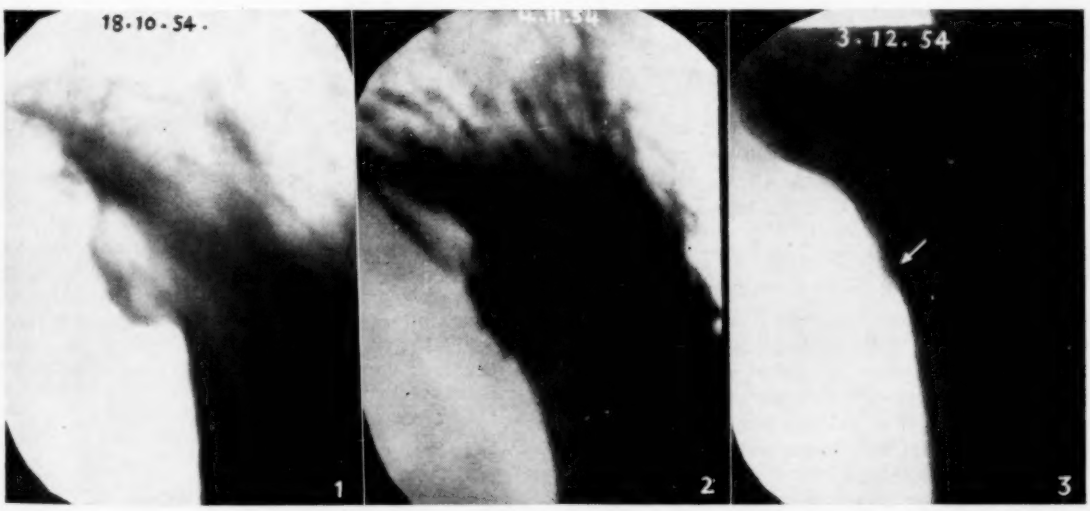
On hormonal, vitamin and belladonna therapy he became symptom-free in 3 days, and achieved complete radiological healing in 3 weeks, at which stage resting blood-sugar was normal. The tolerance curve was not repeated but clinically all symptoms of hypoglycaemia had disappeared.

In view of the constantly reiterated warnings that ACTH and cortisone were absolutely contra-indicated for therapeutic purposes in patients suffering from peptic ulcer, and in view of the success attained with this agent plus vitamin C in Case 2, it was decided to seek a suitable subject and repeat the therapeutic test.

Case 5. *Gastric Ulcer*

E.O.M., aged 57, gave a history of symptoms suggestive of gastric ulcer which had been present for some weeks and were steadily getting worse. X-ray examination on 19 October 1954 (Fig. 1) was reported as follows: 'The crater of a large ulcer was present at a high level on the lesser curvature posteriorly. No direct evidence of any local malignancy was seen but this is impossible to exclude in an ulcer situated at this site.'

He was admitted to a nursing home because of suspected imminence of perforation, and was given 40 units of Acton Prolongatum (slow absorption ACTH) intramuscularly daily and 1,000 mg. of vitamin C intravenously. After 3 days Acton was reduced to 20 units and this was then given every 2nd day. Vitamin continued to be given daily and other therapy comprised 10 mm. of tincture of belladonna *t.i.d.*, 15 gr. of potassium citrate *t.i.d.*, and for the first week an occasional teaspoon of Novasorb. Diet consisted



Case 5. Skiagrams of ulcer. *Fig.* 1. 18 October, *Fig.* 2. 4 November, Fig. 3. 3 December.

of white meats, eggs, cheese, soft vegetables, fruit and milk foods. Three good meals were taken daily and milk or citrus drinks in between.

Another X-ray examination was made on 4 November (Fig. 2) in order to determine as early as possible whether malignancy was involved or not. The ulcer was seen to be reduced to half its original expanse and depth, a most gratifying finding.

Two weeks later the patient was allowed home and he then came in to the consulting rooms regularly for continuance of therapy. On 4 December 1954 further radiological examination was carried out (Fig. 3). The report was as follows: 'The gastric ulcer previously demonstrated at a high level on the lesser curvature posteriorly was still present, but healing appeared almost complete.'

Subsequent progress has been uneventful and the perfect result has been attained. The patient is symptom-free and actively employed, and maintenance therapy consists of 500 mg. of vitamin C by mouth daily in divided doses. Vitamin C remains our sole follow-up insurance against relapse.

ACTH may, in itself, be dangerous to victims of peptic ulceration. Combined with liberal dosage of vitamin C as indicated by Ungar it would at least seem to satisfy the therapeutic requirements of gastric ulcer and may do so for uncomplicated duodenal ulcer. At the same time one's impression is that natural hormones are more effective than ACTH for duodenal ulcers, which is only another indication of the impression one has that these ulcers and the gastric variety are representative of two distinctly different pathologies. What has emerged from this study is that under no circumstances should we capitulate in the face of the ulcer assault. A condition which so consistently reproduces its evolutionary picture should lend itself to solution.

Assuming as correct that the causative background is stress and its multitudinous repercussions, anti-stress agents should supply the therapeutic answer. The first stage towards success is that we must familiarize ourselves with the background, and the disruption and distortion of metabolic harmony which is so characteristic of defence failure. Only by so doing may we position ourselves and organize our curative attack on a scientific basis.

Recently (December 1954) Selye[21] has stated: 'In my opinion research on stress will be most fruitful if it is guided by the theory that we must learn to imitate—and if necessary to correct and complement—the body's own autopharmacological efforts to combat the stress factor in disease.' This is a declaration of therapeutic prophecy which merits our whole-hearted support and approval and embodies principles which it has been one's earnest endeavour to exploit in practical therapy for the past 20 years.

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CLINICAL DIPHTHERIA IN THE NON-EUROPEAN

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Diphtheria is endemic in South Africa, a fact to which Bokkenheuser and Heymann [1] recently drew attention. In view of the high incidence of the disease, an assessment of its present clinical status in the non-European was considered worthwhile.

Material. This study is based on 1,135 cases of diphtheria (corrected for diagnosis) admitted to Waterval Hospital, the non-European Isolation Hospital of the Johannesburg Municipality, during the 3 years 1952-1954. The cases consisted of 1,017 Bantu, 75 Coloured, 41 Indians and 2 Chinese. Practically every case was personally seen by the author, and every bed-letter reconsidered in order to achieve uniformity at least in the diagnosis and classification of the cases presented.

Seasonal Incidence. During the months December to May 712 cases or 62·7% of the cases were admitted. There were most admissions in January and February. This appears a constant feature in this country.

Sex Distribution. The cases comprised 631 females and 504 males. The slightly greater incidence of the disease amongst females appears to be universal.

Age Distribution is shown in Table I. The proportion of cases under the age of one year, viz. 6·4%, is more than 3 times as great as in the London Fever Hospitals in the years 1895-1914,[2] where it was 1·8%. The figures are probably comparable in that they are both taken from a non-immunized population, viz. that existing in

TABLE I. AGE DISTRIBUTION

Age (years)	*No.*	%		*Age (years)*	*No.*	%
Under 1	72	6·4		7-11	223	19·6
1-3	199	17·5	45·6%	11-16	98	8·6
3-5	246	21·7		16 and over	106	9·3
5-7	191	16·8		Total	1,135	99·9

London before the institution of active immunization, and our relatively non-immunized Bantu population. The proportion of cases in adults is nearly the same (10%) in the two series.

Almost half the cases (45·6%) occurred under the age of 5 years, the under-school age-groups. The London figure was 41·7%.

The youngest case was one of nasal diphtheria in a 12-day-old infant with a history of 7 days. The youngest case of faucial diphtheria was in an infant of 6 weeks; the oldest was in a female of 60 years.

History. To obtain an accurate history from an African adult is notoriously difficult; from an unaccompanied baby it is impossible. However, information was obtained in about 340 of the cases. The average duration of illness before admission to hospital was 4 days. Complaints in children were: feeling off-colour, refusal of feeds, some feverishness and often a sharp dry cough. Sore throat was only rarely mentioned, and the necessity of examining the throat regularly in a sick child cannot be over-emphasized. Adults invariably complained of a sore throat and slight constitutional disturbances. Pain, however, was usually not severe and had frequently disappeared even when membrane was still present.

The Type and Severity of Disease is shown in Table II. The types of diphtheria were classified as (1) nasal, (2) faucial, (3) laryngeal, (4) nasal and faucial, (5) faucial and laryngeal, (6) nasal, faucial and laryngeal, and (7) others (cutaneous, aural, conjunctival and vaginal).

TABLE II. TYPE AND SEVERITY

Type	*No.*	%	*Grade*	*No.*	%
Nasal	40	3·6	1st ..	343	30·2
Faucial	887	78·1	2nd ..	390	34·4
Laryngeal	20	1·8	3rd ..	402	35·4
Nasal and Faucial	95	8·3			
Faucial and Laryngeal	80	7·1			
Nasal, Faucial and Laryngeal ..	12	1·1			
Others	1	0·1			

Faucial diphtheria (alone) comprised nearly 80% of the cases, nasal diphtheria (alone) 3·5% (40 cases) and primary laryngeal diphtheria 1·8% (20 cases).

The 'other' types were extremely rare (a single case of cutaneous diphtheria); although ulcers secondarily infected from a virulent nasal discharge, and a case of purulent conjunctivitis secondarily infected in a similar way, did occur. There were no true cases of aural diphtheria, i.e., with membrane in the ear, but there were cases of chronic otitis harbouring virulent organisms in their aural discharges.

The severity of the disease was classified in 3 grades, corresponding to mild, moderate, severe, by a scheme which is a slight modification of Banks'[3]:

Grade 1=membrane on one tonsil, or a small amount on both.

Grade 2=a larger amount on both tonsils, with (*a*) slight involvement only of uvula and pharynx or (*b*) an associated nasal or laryngeal involvement, and with cervical lymphadenitis present.

Grade 3=(*a*) membrane involving tonsils, uvula and pharynx, with an obvious peri-adenitis (bull neck), unilateral or bilateral, or (*b*) a severely toxic case, irrespective of the amount of membrane (the membrane may already have partially stripped by the time the case is admitted).

The 3 grades were more or less evenly represented, with slightly fewer mild cases. Grade 1 would comprise a higher proportion if all early cases were recognized and admitted to hospital.

Bacteriological proof was obtained in a disappointingly small percentage of cases (50%). Apart from faulty technique in taking the swabs and the delay in bacteriological examination (steps have since been taken to rectify these, with a significant improvement in results) the other factor responsible is the liberal use of antibiotics before admission. A patient not infrequently has been treated as a follicular tonsillitis with penicillin for a few days until diphtheria is suspected, by which time the chances of obtaining a swab positive for diphtheria have become slender. The warning must, therefore, be repeated that the diagnosis must not depend upon the bacteriological report. A positive result will confirm, but its absence will not negate the diagnosis. The onus rests primarily on the clinician, but closer collaboration with the bacteriologist will achieve better results.

The Mortality Rate is shown in Table III. While in many other parts of the world the mortality rate has fallen to between 3-5%, it was 12·3% in this series. This is undoubtedly due to the delay in seeking medical attention. Particularly is this the case with admissions from the peri-urban areas. Of the 255 cases from outside

TABLE III. MORTALITY RATE ACCORDING TO AGE

Age (years)	*Cases*	*Died*	%
Under 1	72	13	18·1
1-3	199	34	17·1
3-5	246	39	15·9
5-7	191	23	12·0
7-11	223	26	11·2
11-16	98	4	4·1
16 and over	106	1	0·9
Total	1,135	140	12·3

Johannesburg 58 died, a mortality of 22·7%, while of 880 local cases 82 or 9·3% died. The mortality is shown separately for each age-group. It is highest in the youngest. There follows a steady decrement with each succeeding age-group until the age of 11 years, when the decline is sharp. Only one adult succumbed (a woman), of toxic myocarditis 8 days after admission.

Time of Death. Exactly half the deaths, 70 out of 140, occurred within 48 hours of admission, and a further 64 (45·7%) between the 3rd and 14th days. No deaths took place later than that, until after the 35th day when 6 (4·3%) died.

Cause of Death. An acute toxic myocarditis accounted for 120 (85·7%) of the deaths, and an associated broncho-pneumonia for 9 deaths. Paralysis associated with a late carditis and broncho-pneumonia caused 6 deaths, laryngeal obstruction complicated by a broncho-

pneumonia or a toxic myocarditis 3 deaths, and miliary tuberculosis and gastro-enteritis 1 death each.

DIFFERENTIAL DIAGNOSIS

1. *Nasal Diphtheria.* Two cases of a non-specific rhinorrhoea were encountered. Other conditions to bear in mind are (*a*) congenital lues, (*b*) foreign body, and (*c*) trauma to the septum from constant nose-picking. (Occurring by itself, the condition is not serious and no deaths resulted, but it is a common source of infection.)

2. *Faucial Diphtheria*

(*a*) *Tonsilitis* (254 *cases*). This is the commonest error of diagnosis. In the event of any doubt, the case should be treated as diphtheria. The late Dr. Bayer used to teach that the mere reaching for a throat swab was sufficient indication for the administration of antitoxin.

(*b*) *Stomatitis, including Thrush.* Twenty-two cases were mistakenly admitted as diphtheria. The exudates of thrush are more linear, milky white in appearance, and more diffuse in their distribution in the oral cavity. A stomatitis is also more diffuse; it affects the gums, buccal mucosa and lips and is often associated with a glossitis.

(*c*) *Syphilitic lesions* accounted for 5 admissions. They were all tertiary—2 cases where the palate was undergoing perforation, 2 with secondarily infected congenital perforations, and 1 with syphilitic granuloma extending down to the vocal cords.

(*d*) *Tuberculous ulceration* was present in 5 cases, in one of which it was associated with a fiery red pharynx, resembling an acute streptococcal throat, and in the others with pseudo-membranous exudates. All had a high fever and the diagnosis was established radiologically and by sputum examination. All responded rapidly to streptomycin, although the pulmonary lesions require prolonged treatment.

(*e*) *Tuberculous cervical lymphadenitis* was mistaken for a bull neck in 1 case.

(*f*) A case with post-nasal drip with an exudate on the posterior pharyngeal wall posed a diagnostic problem until an E.N.T. consultant established the presence of *carcinoma of the antrum.*

3. *Laryngeal Diphtheria*

Secondary laryngeal diphtheria presents no diagnostic difficulties. The presence of membrane in the throat is almost conclusive. The diagnosis of a primary laryngeal diphtheria is not always easy.

(*a*) *Bacterial laryngitis or acute laryngo-tracheo-bronchitis* accounted for 64 cases, 8 of which required tracheotomy. It is usually accompanied by high fever and there may be some pharyngeal inflammation. Until proved otherwise, it should be treated as diphtheritic. The mortality for those undergoing tracheotomy is high, owing to the extension of the inflammatory process to the bronchi and bronchioles.

(*b*) *Broncho-pneumonia* (23 cases) was another common source of error, but unlike the laryngo-tracheitis, differentiation is possible and tracheotomy contra-indicated. Dyspnoea, hoarseness, stridor and restlessness are signs shared with diphtheria but, in broncho-pneumonia, fever is usually higher, respiration is more rapid and shallow, and examination of the chest usually reveals bronchial breathing or crepitations.

(*c*) Laryngitis is frequently associated with *measles* (20 cases), and may occur before the appearance of the rash, in which event confusion with laryngeal diphtheria is possible. A laryngitis occurring during the convalescent stage of measles is not infrequently diphtheritic. The association is an accepted entity. There were 6 cases of measles and laryngeal diphtheria in this series. Tracheotomy may be required in either condition.

(*d*) *Miliary tuberculosis.* One case was admitted, the mechanism of the dyspnoea and chest recession being similar to that in broncho-pneumonia.

(*e*) A *mediastinal mass* (1 case), subsequently proved to be leukaemic, was found in a case referred to us as one of laryngeal obstruction.

(*f*) A *congenital heart lesion*, because of dyspnoea and cyanosis, also gained admission for a case as one of laryngeal diphtheria.

(*g*) *A case with massive atelectasis of the left lung*, due to an obstruction of the left main bronchus by a tumour, also presented with dyspnoea and stridor and produced a suspicion of diphtheria. The effect was similar to that of a foreign body, for which one must be on guard, although no cases were discovered in this series.

A retropharyngeal abscess may also produce stridor and be mistaken for laryngeal diphtheria.

LARYNGEAL DIPHTHERIA

Bayer,[4] reviewing the European cases at the Johannesburg Fever Hospital 1930-37, found 1,291 cases of diphtheria, of which 242 (18·7%) were laryngeal and 124 required tracheotomy. In the present series there were 112 cases of laryngeal diphtheria (9·9%), a considerably lower percentage, but the proportion requiring tracheotomy was slightly higher (69 cases—61·6% as against 51·2%). Of the 112, 20 were primary and 92 secondary laryngeal diphtheria.

The *Age Incidence and Mortality of Tracheotomy* is shown in Table IV. Four-fifths of the tracheotomies were performed on children under the age of 5 years. At ages above that, operation was not frequently required, and never over the age of 11. The mortality was 34·8%, which compares favourably with Bayer's figure of 33·3%. Mortality did not appear to be influenced by the age of the patient. This is contrary to Bayer's assertion that 'in children under 1 year the outlook is usually hopeless'.[4] Of 10 cases in this series

TABLE IV. TRACHEOTOMY CASES AND MORTALITY ACCORDING TO AGE

Age (years)	*Cases*	*Died*	
Under 1	10	3	
1-3	25	8	
3-5	21	5	
5-7	8	5	
7-11	5	3	
Total	69	24	(34·8%)

under the age of 1 year, 7 managed to survive. This may be due to the prevention and treatment of the complication of broncho-pneumonia by antibiotics, which were not yet discovered in 1937.

Of the 69 tracheotomies, 30 were performed on bull-neck diphtheria. In spite of the severity of their illness, almost half—14 cases—survived. Laryngeal obstruction must be relieved; extreme toxicity is no contra-indication for operation.

The operation favoured at Waterval Hospital is a low tracheotomy done under local anaesthetic.

COMPLICATIONS OF DIPHTHERIA

The toxins have a specific action on the heart, kidney, and nerve tissue, producing respectively a carditis, nephritis and neuritis.

Carditis

This was the most important complication. It probably occurs even more frequently than can be detected clinically or with an electrocardiograph.

Signs detected clinically were: tachycardia or bradycardia, extrasystoles, a weak first heart sound followed by splitting of the sounds, a systolic murmur, gallop rhythm.

E.C.G. changes were: slurring of the QRS complex, depressed S-T segments, inverted T waves, and bizarre pictures of conduction defects and bundle-branch block.

Prognosis. The majority of deaths were due to peripheral circulatory failure, associated with an acute toxic myocarditis. These deaths took place before the 14th day, but with the onset of paralytic complications there was a recurrence of signs (late carditis).

A gallop rhythm is of prognostic significance. Of 25 cases in whom a gallop rhythm was recorded, 9 died early on of an acute toxic myocarditis, while 13 developed palatal or pharyngeal paralysis, of whom a further 3 died. Half the cases with a gallop rhythm therefore died, and only 3 of the survivors did not suffer further complications.

A marked bradycardia, due to heart block, where the rate dropped to below 60/min, was also usually fatal.

Congestive cardiac failure developed in 5 cases, the earliest 5 days after admission, the latest 37 days. There were 2 deaths, one on the 8th day, the other on the 40th, the latter following the onset of a pharyngeal paralysis. Digitalis was used in 3 cases, 2 of whom recovered. The death was in a case which emerged successfully from congestive cardiac failure occurring on the 18th day, only to suffer a relapse on the 40th day, coincident with pharyngeal paralysis. Digitalis is not indicated in the arrhythmias of diphtheria, but its judicious use in a case with progressive congestive cardiac failure is occasionally life saving.

Renal Complications

Albuminuria, the presenting feature of toxic nephritis, was found in 114 cases (10%). A number of cases died before a urine examination could be made. The presence of albuminuria is an indication of the severity of the illness. Forty-four (38.6%) of those with albuminuria died, and of the survivors a further 36 developed paralysis, with 4 deaths amongst them. Only 1 out of every 3 cases with an albuminuria, therefore, escaped death or other severe complications.

Oliguria and anuria were usually due to peripheral circulatory failure, anuria invariably having a fatal termination.

Neuritis

Ocular and facial palsies were each only noted in 2 cases and in themselves were not serious. Palatal paralysis occurred in 70 cases, an incidence of 6·2%. Twenty went on to develop pharyngeal paralysis, 5 of whom progressed to respiratory paralysis. Three cases required an artificial respirator (iron lung), with 1 death. Of the 70 cases, 6 died (a mortality of only 8·6%), 5 of an associated broncho-pneumonia and toxic myocarditis, while the 6th contracted an acute miliary tuberculosis, which proved fatal.

Some of the cases with palatal, and all with pharyngeal paralysis had a generalized peripheral neuritis, the lower limbs being affected more than the upper. Though convalescence was protracted, recovery was invariable and complete (excluding, of course, the 6 deaths.)

Time of Onset. The earliest onset of a palatal paralysis was the 8th day after admission, and the latest the 50th. The average was the 34th day. Pharyngeal and respiratory paralysis occurred in quick succession.

Age Distribution. No case of paralysis occurred under the age of 1 year, and only 1 in an adult. It was the older children who were most commonly affected.

Bull Neck

This is being considered under a separate heading in order to assess the prognosis. There were 296 cases classified as bull-neck (26·1% of admissions) with 108 deaths, a mortality of 36·2%. Paralytic complications occurred in 55 cases (18·6%), including 5 who died. In summary, 1 in every 4 admissions was a bull-neck case. Of every 6 bull-neck cases, 2 died, 1 developed paralysis, and 3 suffered no further complications. For non-bull-neck cases of grade-3 severity the figures were slightly lower, the mortality rate being 28·3% and paralytic complications 13·2%.

TREATMENT OF DIPHTHERIA

Since the introduction of antitoxin no great strides have been made in the treatment of this preventable disease. The dose of antitoxin used varies from 40,000 to 120,000 units according to the severity of the disease, irrespective of the age of the patient. It is given in one dose and is only repeated if, on reassessment, it appears that the severity was originally under-estimated. Reactions to serum are very rarely encountered in the non-European.

In this series, ACTH and penicillin were used as adjuncts, but one cannot say that the mortality was appreciably influenced. Their use is nevertheless recommended.

In 1952, vitamin B 12 was tried prophylactically in an attempt to reduce the number of paralytic complications, and therapeutically once paralysis had already developed. The results did not appear sufficiently encouraging to

warrant a large-scale trial, and its use was subsequently abandoned.

The arrival on the scene, however, of erythromycin has been an advance in the treatment of the persistently positive case or carrier. Penicillin, aureomycin and terramycin had only moderate success in eliminating the diphtheria bacillus. Blute[5] reported the use of erythromycin in 3 acute cases and 1 carrier, with the immediate disappearance of the bacilli. Since June 1954 we have used it in 20 cases who had persistently positive swabs despite the use of other antibiotics. In every case negative swabs were returned in 24 hours and continued negative.*

PROGNOSIS

Once the toxin has become fixed to the cells, little can be done to influence the course of the disease, but by careful assessment the probable complications can be anticipated. On admission the case is classified according to severity. In grade 1, no complications need be expected. In grade 2, less than 1% will not proceed uneventfully (1 death, and 2 with paralysis out of 390 cases). In grade 3 the chances of a bull-neck case are only slightly graver than those of non-bull-neck cases. One-third of the cases will die, and of the survivors about one-quarter will develop further complications. If a gallop rhythm or an albuminuria are present, paralytic complications can be expected to follow. If the case survives the 14th day, it stands a good chance of recovery. The next danger period is in the 5th week, with the onset of a palatal paralysis, often heralded by a spike in temperature or a listless appearance in a child who has previously been cheerful and playful. Between the acute and paralytic phases the patient often appears quite well, but the prognosis must remain guarded. If palatal paralysis clears before the development of a pharyngeal paralysis, no further complications are likely to follow. If the patient survives, recovery is complete.

* Subsequently we have experienced 2 cases in whom there was a delay of 72 hours after using erythromycin.

CONCLUSION

The tragedy is the large number of cases which made the writing of this article possible. In most other countries, the scourge of diphtheria has disappeared. In South Africa the battle scarcely seems to have begun. The answer is not in finding a better therapeutic agent; the answer is in prophylaxis. The weapon has been in our hands now for many years, but active immunization is still not practised on a sufficiently large scale. Smallpox has practically been eliminated through compulsory vaccination. Similar legislation is required to enforce immunization of all infants against diphtheria.

SUMMARY

1,135 cases of diphtheria in the non-European are presented, with a case mortality of 12·3%, decreasing with age. The differential diagnosis of each type of diphtheria, and the incidence and prognosis of the major complications, are discussed. Mention is made of the use of erythromycin in eliminating the diphtheria bacillus from a case or carrier. The high incidence of the disease is stressed, and a plea is made for legislation enforcing immunization.

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NEPHRITIS IN THE BANTU

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Clinical patterns of nephritis are commonly encountered in Bantu patients (Hennesey, Gelfand). Literature on the subject, however, is scanty, and much of what is written is difficult to interpret in the light of the more modern classifications of nephritis.

At the present time the classification suggested by Ellis (1942), is regarded as being the most practical, with the separation of nephritis cases into types I and II. It is important to realize when applying this classification that although the two types appear clinically and pathologically as different entities they are not entirely separate diseases (Ellis, 1949). Mixed types are encountered, and not infrequently there is a transition of type II into type I (Davson and Platt, Allen, Enticknap and Joiner).

An analysis of the records of 220 cases of nephritis in Bantu patients admitted to the Baragwanath Non-European Hospital during the 5-year period from 1 January 1948 to 31 December 1952 was compared with the records of 168 cases of nephritis in European patients admitted to the Johannesburg Hospital and the Transvaal Memorial Hospital for Children over the same period. (The Baragwanath Hospital caters for non-European patients of all ages; the Johannesburg Hospital for European patients over the age of 14 years and the Transvaal Memorial Hospital for European children under the age of 14 years. The admission rates for European and non-European patients at these hospitals are sufficiently similar to allow of comparison.)

Only the earlier stages of nephritis are considered. Inherent difficulties exist in obtaining early records of cases of the chronic forms of nephritis (the Baragwanath Non-European Hospital was only instituted in 1948). This is obviously an important aspect still to be covered. Pathological confirmation is very necessary in any study of renal disease. Little is available for the present series, for the mortality is low in the early stages of nephritis. This confirmation may only be obtained in years to come.

Classification

Using the criteria of Ellis (1942), the cases were classified into nephritis of types I and II as shown in Table I.

TABLE I. CLASSIFICATION OF CASES OF NEPHRITIS INTO TYPE I AND TYPE II

	Total	*Type-I Nephritis*	*Type-II Nephritis*
European	168	126 (75%)	42 (25%)
Bantu	220	150 (68%)	70 (32%)

The few cases encountered that were considered to be examples of a mixed form of nephritis were classified under what was regarded as the predominant type of nephritis in the particular case.

Age Incidence

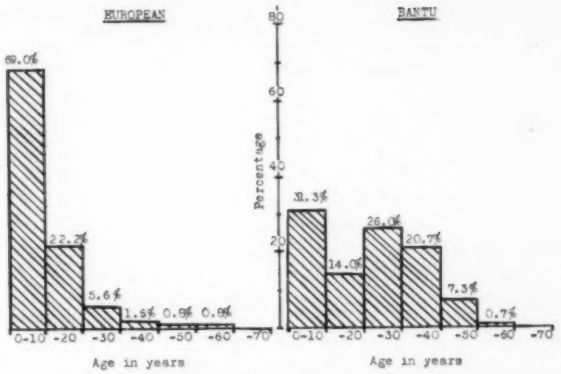
In order to compare the age of onset of the two types of nephritis in the European and the Bantu patients, the frequency distribution of the cases was determined in 10-year age-groups and is set out in Table II and illustrated by means of histograms in Figs. 1 to 4.

TABLE II. DISTRIBUTION OF THE TYPE-I AND TYPE-II NEPHRITIS CASES IN 10-YEAR AGE-GROUPS (AGE OF ONSET OF DISEASE)

Age (years)	*European*		*Bantu*	
	Type I	*Type II*	*Type I*	*Type II*
0–10	87 (69·0%)	24 (57·1%)	47 (31·3%)	—
11–20	28 (22·2%)	5 (11·9%)	21 (14·0%)	18 (25·7%)
21–30	7 (5·6%)	4 (9·5%)	39 (26·0%)	40 (57·1%)
31–40	2 (1·6%)	4 (9·5%)	31 (20·7%)	11 (15·7%)
41–50	1 (0·8%)	3 (7·2%)	11 (7·3%)	1 (1·5%)
51–60	1 (0·8%)	1 (2·4%)	1 (0·7%)	—
61–70	—	1 (2·4%)	—	—
Total	126 (100%)	42 (100%)	150 (100%)	70 (100%)
Average age	10 years	15 years	21 years	27 years

The onset of type-I nephritis in the European patients is at its maximum in the first 2 decades (91·2%). This contrasts with the Bantu, in whom the disease shows a relatively more even distribution over the age-groups up to the end of the 4th decade (Figs. 1 and 2).

The type-II patients of the two races also show a difference in the age of onset of the disease. The European patients have a maximum incidence in the 1st decade (57·1%), and a low incidence of cases occurs at all ages up to the end of the 7th decade. In the Bantu patients there were no cases with onset of the disease during the 1st decade. Almost all the cases began their illness between the ages of 11 and 40 years, with a maximum incidence during the 3rd decade (57·1%). Thus it appears that type-II nephritis is predominently a disease of infants in the European, and a disease of young adults in the Bantu. (Figs. 3 and 4).

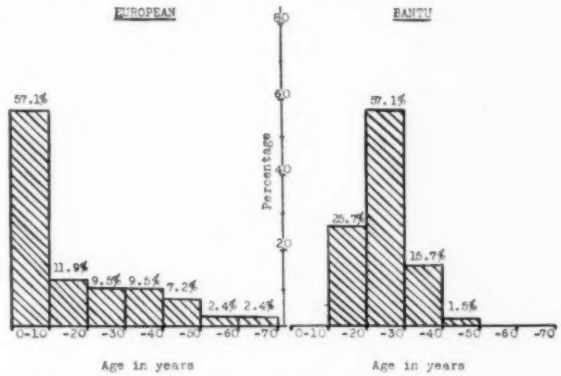


Figs. 1 *and* 2. Type I. Histograms showing percentage distribution in 10-year age-groups of 126 European and 150 Bantu cases of type-I nephritis (age of onset of disease).

Sex Incidence

The sex incidence of the cases is set out in Table III.

The admission rate of male patients to the Baragwanath Hospital is much greater than that of females, owing to the larger male Bantu population in the Johannesburg area. This might account for the greater percentage of male cases of both types of nephritis in the Bantu as compared to the European.



Figs. 3 *and* 4. Type II. Histograms showing percentage distribution in 10-year age-groups of 42 European and 70 Bantu cases of type-II nephritis (age of onset of disease).

TABLE III. SEX INCIDENCE OF TYPE-I AND TYPE-II NEPHRITIS

	European		*Bantu*	
	Type I	*Type II*	*Type I*	*Type II*
Males ..	80 (63%)	16 (38%)	106 (71%)	39 (56%)
Females	46 (37%)	26 (62%)	44 (29%)	31 (44%)
Total ..	126	42	150	70

Mortality Rates

Four of the Bantu type-I cases (2·7%) died in the acute phase, and 7 of the type-II cases (10·0%) died while in hospital with oedema.

Three of the European type-I cases (2·4%) and 10 of the type-II cases (23·8%) died in the acute phase.

The pathology is not reviewed at this stage; further analysis is still necessary.

Most of the deaths in the European type-II cases occurred in children under the age of 4 years. In these children type-II nephritis appears to be a more serious disease than that seen in the young Bantu adult.

CASE REPORTS

The following case histories are all of Bantu patients, and are included to illustrate some of the points mentioned in the discussion.

Case 1

J.M., male aged 33 years. Admitted 3 June 1953.

Complaint. Swelling of whole body for 4 days. Slight breathlessness on exertion for same period. Previously quite well. No sore throats, and no haematuria noted.

Examination. Patient lying comfortably in bed. Temperature 97° F., pulse rate 68 per minute, respiration rate 20 per minute. Generalized oedema involving face, trunk and limbs. Nasal and pharyngeal mucosa congested. Heart not enlarged, sounds normal. Blood pressure, 180/100 mm. Hg. Lungs, abdomen, and central nervous system including fundi, normal.

Urine. Clear, amber-coloured. Specific gravity 1020. Albumin present, 0·5 g. per 100 ml. Microscopic examination of the centrifuged deposit showed moderate numbers of erythrocytes together with a few polymorphonuclear leucocytes and hyaline and epithelial casts.

Blood. Blood urea 47 mg., plasma cholesterol 205 mg., haemoglobin 16 g. (all per 100 ml.). Leucocyte count 7,900 per c. mm. Serum proteins: total 5·6 g. (albumin 2·3 g., globulin 3·3 g.) per 100 ml. Streptococcal antihaemolysin titre 150 units.

Treatment. Rest in bed on low sodium diet. Procaine penicillin 600,000 units intramuscularly daily for 14 days.

Progress. After one week's stay in hospital the oedema had subsided and could no longer be detected clinically. The blood pressure dropped to 120/90 and remained at that level. The blood-urea level dropped to 23 mg. The amount of urinary albumin diminished steadily and after the 17th day in hospital was no longer detectable, and only an occasional erythrocyte could still be detected on microscopic examination of the urine.

Comment. This is an example of an attack of type-I nephritis, with improvement after a short clinical course. The absence of macroscopic haematuria is in no way unusual.

Case 2

G.M., male aged 33 years. Admitted 28 March 1953.

Complaint. Swelling of legs and abdomen of gradual onset for 2 months. He was able to continue with his work as a farm-labourer until 1 week before admission, when the swelling began to increase more rapidly, and he found it difficult to walk about; also he became increasingly breathless on exertion. Two days before admission he noted haematuria for the first time. There is no history of sore throats.

Examination. Patient lying flat in bed, a little dyspnoeic and unable to sit up because of marked abdominal distention. Moderate oedema of legs and over sacrum. Temperature 98° F., pulse rate 90 per minute, respiration rate 30 per minute. Heart not clinically enlarged, sounds normal. Blood pressure 122/80 mm. Hg. Dullness on percussion, diminished air entry, and a few fine crepitations were present at both lung bases posteriorly. X-ray appearance of heart and lungs normal. The abdomen was tensely distended with ascites. The nervous system and fundi were normal.

Urine. Frank haematuria was present on admission.

Paracentesis of the abdomen was performed on the day of admission, and 7 litres of amber-coloured fluid withdrawn. The fluid had a specific gravity of 1010 and contained 1·4 g. of protein per 100 ml. After the paracentesis a smooth soft non-tender liver edge was palpable at the level of the costal margin.

Blood. Blood urea 72 mg., blood cholesterol 186 mg., haemoglobin 11·1 g. (all per 100 ml.). Serum proteins: total 3·9 g. (albumin 0·7 g., globulin 3·2 g.) per 100 ml.

In view of the disproportionately marked ascites, investigations were carried out to determine if liver disease and portal hypertension were present as well as nephritis. Rectal examination revealed no haemorrhoids. Fluoroscopy with barium swallow failed to demonstrate oesophageal varices.

Liver Function Tests. Thymol tubidity 1·0 unit. Thymol flocculation test—negative. Cephalin flocculation test—negative. Takata-Ara reaction—++++. Alkaline phosphatase 7·2 units (King-Armstrong).

Aspiration Liver Biopsy. Showed slight haemosiderosis, and some nuclear irregularity of the parenchymal cells. No evidence of cirrhosis or amyloidosis.

Treatment. Bed rest and salt-free diet.

Progress. The ascitic fluid re-accumulated rapidly and paracentesis was necessary at weekly intervals during the first 5 weeks' stay in hospital. The oedema of the legs and over the sacrum persisted. The macroscopic haematuria subsided rapidly. By the 4th day in hospital red blood-cells were only visible on microscopic examination, together with polymorphonuclear leucocytes and numerous hyaline and granular casts. These microscopic findings and albuminuria (0·5 to 1·0 g. per 100 ml.) persisted throughout the patient's stay in hospital.

The blood-urea level increased steadily until on 4 May, 5 weeks after admission, it had reached 156 mg. and then it began to subside slowly until, on 1 June, it was 38 mg. Paracentesis of the abdomen was found necessary for the last time on 8 May; thereafter the ascitic fluid did not re-accumulate to any appreciable extent, and the oedema of the legs started to diminish.

The patient was discharged from hospital on 15 June, after almost 12 weeks' stay. By this time only minimal oedema of the ankles was present. No ascites could be detected. Blood pressure was 130/80 mm. Hg. Albuminuria (0·5 g. per 100 ml.) was still present and microscopic examination of the urine still showed the presence of erythrocytes and hyaline and granular casts. Blood-urea level was 38 mg.

Comment. This is predominantly a case of type-I nephritis, in which the 'acute' phase has run a rather prolonged course. The oedema and blood urea increased steadily during the first 5 weeks in hospital and then subsided. The persisting of the urinary findings after the oedema and uraema had subsided suggests that the disease remains latent and may progress to chronic (type I) nephritis. The possibility that this was originally type-II nephritis that was converted to type-I nephritis shortly before admission must be considered in view of the history of two months' oedema before admission. As yet we have no explanation for the disproportionately marked ascites in this case.

Case 3

A.M., male aged 36 years. Admitted 3 June 1953.

Complaint. Swelling of body for 3 days. A slight cough 2 weeks before admission, otherwise was quite well until onset of present illness. No sore throats and no haematuria noted. Patient said he was in the habit of consuming about a gallon of kaffir beer a day.

Examination. Lying comfortably in bed. Temperature 98° F., pulse rate 84 per minute, respiration rate 26 per minute. Well-marked oedema of face and limbs and slight oedema detectable in soft tissues over trunk. Parotid glands prominent. Heart was clinically normal. Blood pressure 166/100 mm. Hg. Scattered rhonchi in both lungs. In the abdomen a hard non-tender liver edge was palpable 4 cm. below the costal margin. Free fluid could not be detected clinically. Testes were atrophic.

Urine. Clear, amber-coloured. Specific gravity 1010. Albumin present (0·4 g. per 100 ml.). Microscopic examination of the centrifuged deposit showed moderate numbers of erythrocytes and a few polymorphonuclear leucocytes and hyaline casts.

Blood. Blood urea 72 mg., blood cholesterol 145 mg., haemoglobin 13·7 g. (all per 100 ml.). Serum proteins: total 6·2 g. (albumin 2·4 g., globulin 3·8 g.) per 100 ml.

Liver Function Tests. Thymol turbidity—5 units. Thymol flocculation—++++. Takata-Ara reaction—++. Colloidal-red test—++++++.

Treatment. The patient was restricted to 2 pints of fruit juice daily for the first 4 days. He was then placed on a low-sodium, protein-free diet for a further week; followed by a diet with sodium

restriction only. Intramuscular injection of procaine penicillin 600,000 units daily was given for 10 days.

Progress. The oedema subsided steadily, and was no longer detectable after 1 week in hospital. The urinary findings present on admission persisted; the specific gravity remained constant, in the region of 1010, and did not vary with water loading or water restriction. The blood urea dropped to 46 mg. by the end of the 1st week and to 37 mg. after a further week, but increased to 75 mg. by the 4th week and remained at that level throughout the patient's stay in hospital. The hypertension present on admission persisted with only slight daily variation.

After two months in hospital, the patient's condition remained static, although he was asymptomatic and feeling well, and he was discharged from hospital on 4 July and allowed to return to work.

Comment. This is a case of type-I nephritis that presents the features of chronic renal failure after only a short acute phase, and has most probably progressed rapidly to chronic (type-I) nephritis. This patient contrasts in some respects with the previous one in that although liver disease (probably alcoholic cirrhosis) is present, the oedematous phase is not prolonged and ascites is not a feature.

Case 4

J.N., male aged 28 years. Admitted 24 October 1951.

Complaint. Swelling of legs and abdomen for 2 months. No sore throats or haematuria noted.

Examination. Face puffy, gross anasarca of lower limbs and sacral region. Marked ascites. Heart clinically normal. Blood pressure 115/80 mm. Hg. Dullness and diminished air-entry at both lung-bases posteriorly. X-ray of the chest showed bilateral pleural effusion.

Urine. Heavy albuminuria (1·2 g. per 100 ml.). Microscopic examination of the centrifuged deposit showed a few hyaline and granular casts, with only an occasional erythrocyte.

Blood. Blood urea 30 mg., blood cholesterol 680 mg., haemoglobin 16·3 g. (all per 100 ml.). Serum proteins: total 4·4 g. (albumin 0·2 g., globulin 4·2 g.) per 100 ml.

Treatment. Salt-free high-protein diet.

Progress. The oedema increased steadily despite treatment. Three weeks after admission the patient had a sudden attack of breathlessness associated with a brisk haemoptysis. He suffered repeated small haemoptyses during the following 3 weeks, and died suddenly on 15 December 1951 after another acute attack of dyspnoea followed by circulatory collapse.

Post-mortem examination showed 2 recent infarcts in the left lower lobe of the lungs. The kidneys were large and pale and on microscopic examination showed the typical features of type-II nephritis.

Comment. This is a case of type-II nephritis. Throughout the stay in hospital hypertension was never observed. Blood-urea estimations were carried out repeatedly, and 40 mg. per 100 ml. was the highest recorded. Heavy albuminuria was constantly present.

DISCUSSION

The classification of Bantu cases of nephritis in this series indicates that type-I nephritis is encountered more frequently (68%) than type-II nephritis (32%).

There is a popular misconception that type-I nephritis is rare in the Bantu, and that type II is the more common form of nephritis in these people. (Davidson.)

In the adult Bantu patient with type-I nephritis macroscopic haematuria is uncommon, although microscopic haematuria is almost invariably present at some stage of the disease. The oedematous phase although acute in onset may occasionally last as long as 3-4 months even in cases that eventually make a complete recovery.

The clinical picture of one of these patients when admitted to hospital, often several weeks after the onset of the illness, is superficially similar to that of a patient with type-II nephritis. Temporary hypertension and nitrogen retention are usually noted. These signs are absent as a rule in type-II nephritis except in the terminal stage of the disease.

Low serum-albumin levels are often encountered in patients with either type of nephritis. Liver disease and malnutrition may be contributory factors. It seems unlikely, however, that liver disease and malnutrition are causes of the prolonged oedema in some of the Bantu cases of type-I nephritis (see cases 2 and 3). Very low serum-albumin levels have been noted in patients that have had only a relatively short oedematous phase, and these low levels usually remain unchanged long after the patients have lost their oedema.

On the other hand the course of type-I nephritis in most of the Bantu children of this series appeared to correspond more to the classically accepted pattern; i.e., acute onset of oedema and haematuria following upper respiratory or other infections, often associated with transient hypertension and nitrogen retention, the signs and symptoms usually subsiding fairly rapidly with rest in bed.

It is interesting that the average age of the Bantu patients with type-I nephritis is 21 years, and that the cases are distributed more evenly above and below this age as compared to the European, in whom it is predominantly a disease of young children (Figs. 1 and 2); also that in the Bantu type-II nephritis is more a disease of young adults as compared with the European, in whom it is commonest in children (Figs. 3 and 4).

SUMMARY

An analysis of the records of 220 cases of nephritis in Bantu patients is made and compared with the records of 168 cases in European patients. In the Bantu, type I is the commonest form of nephritis, and occurs with almost equal frequency in children and in adults.

Absence of macroscopic haematuria, and a prolonged oedematous phase, are not uncommon features of type-I nephritis in Bantu patients.

In the Bantu, type-II nephritis is predominantly a disease of young adults.

I should like to thank the Superintendents of the Johannesburg General Hospital, the Transvaal Memorial Hospital for Children, and the Baragwanath Non-European Hospital, for permission to make use of the case records. Special thanks are due to Dr. L. Hirsowitz, Physician at the Baragwanath Hospital, for his help and guidance.

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THE AMERICAN CANCER SOCIETY*

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The main objective of the American Cancer Society is to control cancer through a comprehensive programme, which includes educating the public to seek medical advice at the earliest opportunity, keeping the medical profession informed on current advances in the cancer problem, supporting facilities for early diagnosis and treatment, and assisting cancer research on a nation-wide basis. This programme is supported entirely by public subscriptions, which today exceed 20 million dollars annually.

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2. '*Cancer*', a bi-monthly professional publication, is sent on a complimentary basis to most of the medical schools and hospital libraries in the country.

3. '*Cancer Current Literature*', a monthly listing of current articles on cancer in the world's literature, is indispensable to research workers and clinicians interested in the cancer problem. It is obtainable for a nominal annual fee.

4. The cancer section in *Experta Medica*, a monthly review of digests and abstracts of important articles from the world's literature of cancer, is also sent free to most of the United States medical school and hospital libraries.

Monographs. The Society is producing a series of authoritative monographs on cancers of specific organs, including, e.g., *Cancer of the Oesophagus and the Stomach*, *Cancer of the Lung*, *Carcinoma of the Breast*. These have been distributed free to practically every physician and medical student in the country. A similar monograph on *Mouth Cancer* was prepared for the dental profession.

Lists of medical speakers, motion pictures, lantern slides and prepared exhibits for medical meetings are always available. A series of excellent movie films (many in full colour) on the problem of the early diagnosis of cancer, has been produced with the collaboration of the National Cancer Institute. They include *Breast Cancer*, *Gastro-intestinal Cancer*, *Uterine Cancer*, *Oral Cancer*, *Lung Cancer*, and *Cancer—The Problem of Early Diagnosis*. There is also a film on *Exfoliative Cytology, a Method in Diagnosis*, and two films entitled *What is Cancer?* and *From one Cell*.

Cancer refresher courses are arranged periodically for general practitioners, and opportunities for post-graduate study is also made available through fellowships and traineeships in clinical cancer. Advanced training for the specialist is similarly provided through fellowships in particular fields. Moreover, special grants are available to support worth-while educational projects such as the publication of medical books and laboratory manuals, and the preparation of films.

An important function of the Society is its leadership in the staging of national and local conferences in which leading specialists in particular forms of cancer meet to discuss specific aspects of the disease, or to evaluate latest practical developments. The *Proceedings* of the First and the Second National Cancer Conferences of 1949 and 1952 have been published and form valuable sources of information on many aspects of cancer.

Education of the Public

The Society, as one of its primary objectives, endeavours to provide sufficient knowledge about cancer to everyone so that prompt and efficient action against the disease may be taken at the earliest possible opportunity.

As a means of overcoming ignorance of the facts, misconceptions of the disease, superstitions and unfounded fears, countless pamphlets on various aspects of cancer, or warnings against quack cancer curers, are constantly being distributed. For the same purpose motion pictures are shown, exhibits are displayed in public places, medical speakers are provided for special meetings, and much propaganda is made over the wireless and television stations, or through the newspapers and posters.

The following is a list of excellent motion pictures (mostly in colour) presenting basic facts to the public, while at the same time offering them messages of hope and dispelling fear: *Man Alive*, treats the subject of cancer and the psychology of fear; *The Warning Shadow* on lung cancer; *Traitor Within*, an animated cartoon on the story of cancer; *Living Insurance*, advising regular physical examination for cancer detection; *You, Time and Cancer*, *Time is Life*, and *The Doctor Speaks His Mind*, are all films stressing the necessity for early diagnosis; *Crusade*, the story of the American Cancer Society; *Miracle Money*, showing the activities and exposure of a quack doctor treating cancer; *Breast Self Examination*, for women's groups. All these films are extensively used throughout the country.

Although most of this propaganda is aimed at the adult population in the so-called 'cancer age-group', attempts are also constantly being made to educate the younger generations in the high schools and universities on the basic facts of cancer.

Professional Service to the Public

The American Cancer Society encourages and assists in the establishment of adequate cancer clinics for diagnosis and treatment. Programmes for cancer detection, whether in special clinics or in doctors' surgeries are supported. It does not supply medical or laboratory facilities in cancer, nor does it treat patients.

Owing to the prohibitive costs entailed in equipping and maintaining special Cancer Detection Clinics, the Society is stimulating a nation-wide programme for cancer detection in the doctor's consulting rooms. This is popularly known as 'The Five-Point Cancer Detection Scheme', the slogan of which is: 'Every Doctor's Office a Cancer Detection Centre'.

The Five-Point Cancer Detection Scheme consists of the routine physical examination of presumably well persons, special attention being paid to the following 5 situations: (1) the skin and lymph nodes, (2) the oral cavity, (3) the breasts, (4) the genitalia, and (5) the rectum. It is estimated that by this examination approximately 60% of all cancers should be detected. Participating doctors are encouraged to keep records for statistical purposes on uniform cards supplied for the purpose. The fees for indigent patients are paid by the Society.

Cancer Information Services are maintained by the local Divisions, and these form an essential link in the chain of cancer control. Their function is to provide the public with general information on cancer, including the available local services to which cancer patients can be referred; no medical advice as such is tendered.

Service to Cancer Patients

Inasmuch as they help the cancer sufferers and their families to face the difficult physical, emotional and financial burdens of a long-term illness, *voluntary lay service* to cancer sufferers and their families form an important part of the activities of the American Cancer Society's local divisions. These services include the making and distribution of dressings to domiciliary cancer patients; providing transport to and from hospitals and clinics; maintaining loan and gift centres from which patients can obtain sickroom equipment and comforts; maintaining a home-visitor service through which trained volunteers perform helpful non-

* A continuation of the summary of a report by Dr. Berman published in last weeks' *Journal* (page 570).

professional duties in the homes of cancer sufferers during convalescence; assisting rehabilitation after operations for cancer.

Cancer Research

It is the firm policy of the American Cancer Society to allocate not less than 25% of the collected funds to cancer research. The Society, however, does not own laboratories or conduct research, but serves as an administrator for the money assigned for the purpose.

Applications for grants, fellowships and scholarships are considered and evaluated by the Committee on Growth of the National Research Council of America, upon whose recommendation the necessary funds are made available—usually on an annual basis. Since the exact nature of cancer is unknown, basic research is fostered in many scientific fields, including biology, biochemistry, biophysics, chemotherapy, and in clinical investigations. In some cases the Divisions of the Society also render financial support to local research projects.

Epidemiological studies are conducted to determine the exact conditions under which some cancers are to be found and to discover new factors which may control the disease. The Society also collects accurate statistical data on cancer and renders consultative service in the analysis of such data to research workers in hospitals and elsewhere.

Allocation of Funds

In the annual allotment of the funds collected by the Society, priority is given to educational and research purposes. Only a small proportion is set aside for direct service or financial aid to individual cancer patients.

The contributed funds are usually spent as follows:

25%: In nationally planned cancer research projects.

15%: For the production of educational material (exhibits, journals, pamphlets, books, movies, etc.); fellowships in clinical cancer; library; statistical; administrative; publicity; fund raising; consultative services.

60%: Stays in the Division for its cancer programme.

A typical Budget is that of the Alleghany County Unit of the American Cancer Society in Pittsburgh. During 1953 the collected funds, which far exceed the required quota, totalled $286,362. This money was spent as follows:—

	$
Research: Local and nation-wide projects	71,590
Service: Aids to the Unit's cancer patients and their families, services rendered by local institutions, improvements of services at local treatment centres by purchase of apparatus, etc.	54,383
Education: Provision of the latest information to the general public and the medical profession	52,275
National Headquarters: To help pay for production of films, pamphlets, printed material, etc.	42,954
Pennsylvania Division: Contribution to state-wide programmes in co-operation with the Cancer Commission of the Pennsylvania State Medical Society	28,637
Campaign: To pay for materials, supplies and temporary personnel of the Unit's special appeal	25,783
Administration: Cost of planning and operating all local programmes	10,740
Total	286,362

TRACE-ELEMENT DEFICIENCIES IN PLANTS AND THEIR RELATION TO KWASHIORKOR

K. H. SCHÜTTE, PH.D.

University of Cape Town

The type of protein deficiency in infants called kwashiorkor has been described by Brock and Autret[1] as 'the most serious and widespread nutritional disorder known to medical and nutritional science'. Various aspects of this condition are being widely studied today from the medical point of view. There is a close relationship between malnutrition and the available sources of food, and the dependence of staple vegetable foodstuffs on the constitution of the soil in which they are grown is of particular importance. Brock[2] and other workers in this field have recognized that mineral deficiencies of the soil and of the crops growing on it may have an important bearing upon the health of the people living in such an area. This is especially true in Africa.

Schütte's[3] survey of trace-element deficiencies of plants in Africa clearly showed the very extensive distribution of these deficiencies. In much of Africa the indigenous population lives primarily upon a vegetable diet, which is not supplemented by much animal protein as the diet is in most developed countries.

McLester and Darby's contention, that human beings do not suffer from trace-element deficiencies because they have a constant source of the necessary trace-elements in the animal part of their diet,[4] does not apply to large areas in Africa. Thus plant deficiencies of a serious nature may be of direct importance in the study of human diseases.

Trace-element deficiencies in plants may influence the well-being of man in several ways. Firstly, they can cause a serious decrease in crop yields and promote conditions likely to lead to undernutrition and famine. But they can also alter the chemical nature of the crops. This is not generally appreciated. The nature of plant proteins may be altered and the relative amounts of essential amino acids markedly depressed.[5, 6, 7] The amino-acid content of plant proteins may vary and low concentrations of certain amino acids may limit the efficiency of the utilization of the proteins present in the diet. Auffret and Tanguy,[8] and Bigwood,[9] have stressed the importance of deficiencies of essential amino acids in kwashiorkor, while Popper *et al.*[10] have shown that deficiencies of sulpha-amino acids such as cysteine and methionine can produce liver lesions in rats.

The other important aspect of trace-element deficiencies in African diets is that they may seriously reduce the activity of enzyme systems in the body, especially in the liver. In kwashiorkor,[1, 11, 12, 13, 14] as well as in the livers of rats suffering from protein deficiency, there is an alteration in the enzymatic pattern of the cell and the activity of certain enzyme systems is greatly decreased. In acute cases the enzymatic machinery may be disrupted.

This alteration in the enzyme systems may be due to deficiencies of amino acids. Williams and Elvehjem[15] have shown that methionine concentration can be a limiting factor in xanthine oxidase activity and also that this enzyme may decrease markedly without a decrease in the non-enzyme liver proteins in rats. Further, Westerfeld and Richert[16] have shown that molybdenum appears to be part of this enzyme, and that its concentration will govern the amount of xanthine oxidase present in the liver of rats.

Iron, copper, cobalt and zinc[4] are all essential constituents of important enzyme systems, which obviously cannot function adequately if only subminimal concentrations of these minerals are present. Magnesium[17] is also necessary for proper development, especially in women. As these elements are all frequently and widely deficient in plants, it is very possible that adequate quantities are frequently deficient in the diet of Africans. In infants this is probably accentuated by the fact that the nutritional states of many lactating women is poor.[11,18]

It is thus very probable that trace-element deficiencies in plants is one of the important subsidiary factors contributing to the widespread occurance of kwashiorkor in Africa and that the effect of trace-element supplements in the treatment of this condition may be important.

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ASSOCIATION NEWS : VERENIGINGSNUUS

CLINICAL CASES DISCUSSED AT MEETING OF THE GRIQUALAND WEST BRANCH

At a meeting of the Griqualand West Branch of the Medical Association of South Africa held at the Kimberley Hospital on 26 May 1955, Dr. E. E. Stephens was in the Chair and 20 members attended. Several items of business were discussed.

Dr. H. F. Loewenthal discussed two cases of ureteral transplants in Native females with vesico-vaginal fistula; the first operated on by retroperitoneal approach, the second with a mucosa-mucosa anastomosis by the peritoneal approach. The intention is to re-transplant the ureters in a year's time back into the bladder.

Dr. T. W. Mills demonstrated a man whose foot was mangled in an agricultural machine with multiple fractures. A Symes amputation was done.

The advisability of this method of amputation was discussed from the floor.

Dr. D. E. Stephens discussed a case of neurofibromatosis in a female of 75 with extensive osteitis of tibia, fibula and ankle joint shown by X-ray.

The meeting was closed with a vote of thanks to the Chair.

IN MEMORIAM

HELLMUTH HEINRICH EDUARD SCHULZ, M.A., M.R.C.S. (ENG.), L.R.C.P. (LOND.)

The death took place in Montagu Hospital on 5 May 1955 of Dr. H. H. E. Schulz, aged 59 years, of Ashton, C.P.

Heinrich Eduard Schulz was born in 1896 at Pniel, near Barkly West, and educated at the Grey University College, Bloemfontein. After lecturing for a while in botany and physics at the Potchefstroom University College, he proceeded to study medicine overseas at Leipzig, Amsterdam and finally at St. Thomas' Hospital, London. There he qualified with the conjoint diplomas of the Royal Colleges, and then returned to South Africa. Commencing at Whites, O.F.S., he later practised in the Western Province at Robertson, Porterville and Paarl and finally settled at Ashton.

Dr. H. H. E. Schulz

Dr. Schulz was a keen anthropologist and botanist; he spent many years collecting Dutch remedies and classifying them on a scientific basis. Recently he collaborated with 3 American scientists in studying various species of indigenous South African medica, and an American laboratory has taken over his collection of herbs. The bulk of his anthropological library has been left to the University of the Witwatersrand, and his botanical and art books to the University of the Orange Free State. In 1937 he received the Hamilton Maynard Memorial Medal from the Medical Association of South Africa.

Dr. P. W. J. Keet writes: Dr. Schulz was well known to me over a long period, during which I came into frequent contact with him. He was a man of a peaceful and even temperament, one not easily disquieted or perturbed. He was highly cultured and keenly interested in scientific studies, particularly in physiology, anthropology, ethnology and botany. His extensive library attested to his interest in art, literature and music.

He was *persona grata* with his colleagues, and respected and revered by all who knew him well, not only for his knowledge of medicine and paediatrics, but also for his idealism in his profession. He had the full confidence of his patients, who valued his serene manner, his tactful and sympathetic approach, and his diagnostic ability. Patients came from far afield to consult him.

His death, after a short illness, during which his colleagues at Montagu and Ashton gave him their constant attention, was keenly felt by his patients and friends.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

'*Pentoxylon*' is a new Riker product which provides a novel approach to the problem of angina pectoris. The manufacturers state: Each Pentoxylon tablet contains 10 mg. of pentaerythritol tetranitrate (PETN) with 1 mg. of Rauwiloid. The rationale underlying this combination is to break the vicious cycle of psychophysiologic events, beginning with the tachycardia attendant on pain and apprehension, which in itself is sufficient to augment dangerously the work demand on the myocardium and so increase the severity and frequency of attacks.

The Rauwiloid component allays apprehension and induces a desirable bradycardia; the PETN exerts its usual long-acting coronary vasodilation effect, thus beneficially increasing exercise tolerance and coronary flow.

Published reports state that true objective improvement, as shown electrocardiographically, can be demonstrated in more than 50% of cases; all cases enjoy good subjective improvement. Pentoxylon is available through the usual channels, while detailed literature and clinical trial material can be obtained on application to the distributors, Riker Laboratories Africa (Pty.) Ltd., P.O. Box 1355, Port Elizabeth.

UNIVERSITY OF THE WITWATERSRAND MEDICAL GRADUATE ASSOCIATION

A week-end postgraduate course in Surgery has been arranged by the Department of Surgery to take place on 12, 13 and 14 August. Provisional programme (subject to alteration):

Friday 12 *August*

9—12 a.m. Ward Rounds.
2—5 p.m. 1. Diagnosis and Management of Rectal Bleeding.
2. Late sequelae of Surgery of Peptic Ulcer.
3. Surgery of the Gall Bladder and the Appendix.
8 p.m. Symposium on the Diagnosis of the Acute Abdomen in the Child, Adult and Aged.

Saturday 13 *August*

9—12 a.m. Ward Rounds.
2—5 p.m. Common Orthopaedic Conditions:
1. Backache.
2. Foot and Hand Injuries.
3. Recent Advances in Fractures.
Evening. Cocktail Party.

Sunday 14 *August*

9—12 a.m. 1. Common Urological Problems in General Practice.
2. Indications for the Surgery of Mitral Disease.
3. Result of Surgery of Pulmonary Tuberculosis.
2—5 p.m. Modern Trends in Peripheral, Arterial and Venous Disease.

Fee for the course is £4 4s. 0d. which includes the University Registration of £1 0s. 0d.

All lectures will take place in the Lecture Demonstration Theatre (Room 45), Department of Surgery, Medical School, Johannesburg (unless otherwise stated).

This course is expected to be popular, and so early application is advisable.

PASSING EVENTS : IN DIE VERBYGAAN

Union Department of Health Bulletin. Report for the 8 days ended 2 June 1955.

Plague, Smallpox: Nil.

Typhus Fever, Cape Province: No further cases have been reported from the Xalanga Magisterial district since the notification of 5 May 1955. This area is now regarded as free from infection.

Epidemic Diseases in Other Countries:

Plague: Nil.

Cholera in Akyab (Burma); Calcutta (India).

Smallpox in Moulmein, Rangoon (Burma); Phnom-Penh (Cambodia); Bombay, Calcutta, Delhi, Jodhpur, Lucknow, Nagpur, Tellicherry, Visakhapatnam (India); Tourane (Viêt-Nam); Mogadiscio (Somalia).

Typhus Fever in Alexandria (Egypt).

* * *

Erratum. In the English version of the editorial on 'Bone Remodelling' in the issue of the *Journal* of 4 June 1955 (page 537) a line was omitted destroying the sense of the sentence. The passage should have been as follows: 'In the long bones the external effect of this remodelling is the absorption of outer layers of bone, so as to produce the tapering which occurs as we travel from the epiphysis along the shaft. If it were not so an adult femur, for instance, would be the same width in the shaft as it is at the lower end. Internal remodelling produces the dense secondary trabeculae of the cortex, the spongy cancellous bone of the medulla and the actual marrow cavity.' The Afrikaans version of the article was correct.

BOOK REVIEWS : BOEKRESENSIES

THE CASUALTY DEPARTMENT

The Casualty Department. By T. G. Lowden, M.A., B.M., B.Ch., F.R.C.S. Pp. 278+viii with 170 illustrations. 37s. 6d. Edinburgh: E. & S. Livingstone Ltd. 1955.

Contents: 1. Sepsis and Antibiotics. 2. Common Septic Conditions. 3. The Septic Hand. 4. Skin Conditions. 5. Uncommon Septic Conditions. 6. The Closed Soft-Tissue Injury. 7. Soft-Tissue Injuries with Breach of Surface. 8. Skin Deficiency. 9. Burns and Scalds. 10. The 'Cold Case'. 11. Anaesthesia. 12. Organisation. 13. The Financial and Temperamental Background. 14. Disposal. 15. Legal Responsibilities. 16. Legal Protection. Conclusion. Appendices. Index.

In this book Mr. T. G. Lowden has succeeded in relating his vast experience of managing a Casualty Department in a clear, well-illustrated, concise book.

The author, through careful study and follow-up of cases, has formulated sound treatment for the various minor surgical complaints encountered in the average casualty department. The treatment suggested is based on sound principles, always with the aim in mind of practising correct surgery which will enable the patient to regain maximum function of the diseased or injured part in as short a time as possible. This is of the utmost importance in labourers attending with sepsis of the hand or soft-tissue injuries.

The author strongly deplores the routine use of antibiotics, which are so often administered to 'cover' bad surgery, and aptly points out that 'antibiotics are not intended to make it any *easier* for the surgeon, but are intended to make it *better* for him'.

Useful advice is also given on the organisation of a casualty department and the legal responsibilities of a casualty officer.

The management of a casualty department is often left in the hands of a young doctor with limited surgical experience. This book should act as a useful guide to such doctors, enabling them to practise correct surgery. It will also be of great help to the average general practitioner who is often called upon to deal with minor surgical complaints in his surgery, especially to those practitioners in country towns where the resources of a hospital casualty department are not so readily available.

The book is strongly recommended for students, house surgeons, casualty officers and general practitioners.

J.H.L.

MODERN TRENDS IN OPHTHALOMOGY

Modern Trends in Ophthalmology. Edited by Arnold Sorsby. Third Series. Pp. 346+xiv with 111 illustrations. London: Butterworth & Co. (Publishers), Ltd. S.A. Office, Butterworth & Co. (Africa) Ltd.

Contents: 1. Microscopy. 2. The Measurement of Aqueous Flow in the Experimental Animal. 3. Some Newer Aspects of Binocular Vision. 4. Some Current Biochemical Problems. 5. Optical Aberrations. 6. Diagnostic Criteria of Genetic Affections. 7. Slit-Lamp Microscopy of the Posterior Segment of the Eye. 8. Measurement of Diplopia Fields. 9. Various other Procedures. 10. The Nature of Malformations. 11. Current Aspects of Ocular Pharmacology. 12. Action of X-Rays and Radoactive Substances on Ocular Tissues. 13. Other Aspects. 14. Muco-Cutaneous Ocular Syndromes. 15. Ocular Aspects of Diffuse Collagen Disease. 16. Allergy. 17. Some Newer Clinical Entities. 18. Streptomycin and the Newer Antibiotics. 19. Cortisone and ACTH in Diseases of the Eye. 20. The Treatment of the Virus Diseases. 21. The Treatment of Hypertensive Retinopathy. 22. Present Status of Corneal and Retinal Surgery. 23. Vertical Muscles. 24.

Contact Lens Fitting—Limitations and Difficulties. 25. Some Surgical Technicalities. 26. Blindness in England and Wales. 27. The Visually Handicapped. 28. Trachoma as a Mass Problem. Index.

It is only 8 years since Arnold Sorsby edited the last of this series of Modern Trends in Ophthalmology, but the considerable developments in Ophthalmology during this period have made it necessary to publish again.

This volume is little more than half the size of the earlier two. In just over 300 pages a very wide range of subjects is covered, as indicated in the 28 chapters listed above. It is therefore not suprising that, in spite of the fact that each chapter is written by an expert of international repute, one is left wishing that some of the articles were a little fuller. On the whole, however, one is bound to admit that a great deal of comparatively recent information of a practical as well as theoretical nature has been crammed into these 300 pages. There is a small selected bibliography after each chapter which points to the major original works on each subject being discussed.

In the chapters on Optical Aberrations it is interesting to learn that night myopia to the extent of ·2 diopters can occur in an emmetrope who becomes myopic in the dark.

In Current Aspects of Ocular Pharmacology, the mechanism of action of many of the newer drugs is discussed. Among these, attention is drawn to the fact that eserine, if instilled into an eye before D.F.P., will block the latter's action whereas, if the DFP is instilled first, a combined activity is obtained. In other words, the sequence in which certain drugs are used is important if a synergistic effect is to be obtained.

The use of radio-active isotopes in the localization of intraocular tumours is referred to.

It is interesting to read in sequence one chapter on Abnormalities of the Vertical Muscle by the English authorities, Lyle and Cross, followed immediately by an article on Surgery of the Oblique Muscles by Fink the American, whose fine dissections and technique for oblique surgery have earned him a prominent position in the field of vertical squint.

Other surgical matters of interest are: a brief description by Scheepers of his most useful binocular operating ophthalmoscope for retinal detachment surgery; chapters by Stallard on Corneal Sutures and Corneal Grafting; by Shapland on Lamellar Sclerectomy; by Harold Ridley on Intra-Ocular Acrylic Lens implant; by Frederick Ridley on Contact Lenses; and so forth.

Enough has been said to indicate that this book can be recommended as a worthy addition to one's reference library.

R.L.H.T.

SIDE EFFECTS OF DRUGS

Reactions with Drug Therapy. By Harry L. Alexander, M.D. Pp. 301+xii with 33 illustrations. Philadelphia & London: W.B. Saunders Company. 1955.

Contents: 1. Introduction. 2. Mechanisms. 3. Dermatologic Manifestations. 4. Systemic Patterns. 5. Anti-Infectious Drugs. 6. Anti-Infectious Drugs (continued). 7. Anti-infectious Drugs (continued). 8. Antiarthritic Drugs. 9. Drugs Used in Cardiovascular Disorders. 10. Sedative Drugs. 11. Antithyroid Drugs 12. Antihistamine Drugs. 13. Organ Extracts. 14. Vitamins. 15. Serums and Vaccines. 16. Plant Products. 17. Local Anesthetics. 18. Miscellaneous Drugs. Index.

A great responsibility rests on those who have to deal with drugs; and not only on those who give them to patients, but also on the few whose duty it is to teach scientifically about them. Hardly a week goes by without an account being published of an unusual experience with drug therapy. Most journals carry almost regularly some article indicating the possible side-effects of drugs. The incidence of drug reactions has multiplied in recent years as more and more people are exposed to drugs that can result in sensitization. Serious accidents and numerous fatalities have been reported. The literature dealing with these problems is extensive and widespread. For this reason and others the book under review is most valuable. It presents a concise account of the untoward reactions produced by most modern drugs; poisoning from overdosage is not included, nor such pharmacological reactions as are to be expected. The book deals therefore with 'drug hypersensitivity', and this is considered in detail the different lesions that may be produced by drugs in common use being carefully described, with explanations of the mechanisims involved. Ample illustrations and lists of drugs indicating the relative capacity to produce reactions are provided, and each chapter is documented with numerous references. This is obviously a book that every doctor should read and have available for reference.

N.S.

ADVANCES IN PEDIATRICS

Advances in Pediatrics. Edited by S. Z. Levine. Volume VII. Pp. 351 with illustrations. $8·00. Chicago: Year Book Publishers, Inc. 1955.

Contents: 1. On Fibrous Defects in Cortical Walls of Growing Tubular Bones. 2. The Urinary Tract in Childhood. 3. Malnutrition in Infancy and Childhood, with Special Reference to Kwashiorkor. 4. Phonocardiography in Children. 5. Infantile Cerebral Palsy. 6. Mucoviscidosis. 7. Congenital Megacolon.

This volume contains 7 authoritative monographs on subjects of contemporary paediatric importance. All are well written, concise and up-to-date and this book should appeal not anly to paediatricians but also to all practitioners who are interested in children.

The first article is written by that well-known paediatric radiologist, Caffey, who records the course and variations in development of asymptomatic cortical bone defects in children. This is an original contribution based on serial radiological observations of a large number of cases; the article is profusely illustrated with X-ray plates and some micro-photographs, and the defferential diagnosis is discussed in detail. Then follows a concise but very adequate résumé of paediatric urology with special reference to obstruction and infection, congenital anomalies, and neuromuscular uropathy; treatment is featured and there are excellent plates, diagrams and classification tables for ready reference. In the article on phono-cardiography consideration is given particularly to physiological systolic murmurs, congenital heart anomalies and the heart in rheumatic fever. Perlstein outlines the modern concept of cerebral palsy, its symptomatology and management. The book contains, too, an excellent monograph on the surgical treatment of congenital megacolon.

Two subjects remain, both of nutritional interest: mucovisidosis and malnutrition. The syndrome of mucoviscidosis is fully dealt with and admirably presented. All aspects are considered and emphasis is placed on unusual clinical features, conflicting data, laboratory diagnosis, and therapy. An interesting observation is that evidence is accumulating which suggests that it may be possible to make the diagnosis on the basis of sweat electrolyte abnormality. South African practitioners will no doubt be especially interested in the article on malnutrition and kwashiorkor. Gomez and his co-workers review all aspects of these conditions and contribute their observations on the clinical picture, pathology and biochemistry of malnutrition based on a large number of Mexican cases. These authors refer to a 'nutritional recovery syndrome' which apparently appears about 3 weeks after treatment has commenced. Not everyone will agree, however, with the dictum that the term 'kwashiorkor', if used at all, should be limited to African (Native) malnutrition cases with skin and hair changes, or with the statement that supplementary vitamin therapy is entirely without benefit in malnutrition.

W.E.

PSYCHO-ANALYSIS

The Technique of Psycho-Analysis. By Edward Glover, M.D. Pp. 404+x. 35s. London: Baillière, Tindall & Cox. 1955.

Contents: Part I. 1. Preamble to Analysis. 2. The Opening Phase (1). 3. The Opening phase (2). 4. Defence-Resistance (1). 5. Defence-Resistance (2). 6. Counter-Resistance and Counter-Transference. 7. The Transference-Neurosis (1). 8. The Transference-Neurosis (2). 9. The Terminal Phase (1). 10. The Terminal Phase (2). 11. Active Technique. 12. The Analyst's Case-List (1). 13. The Analyst's Case-List (2). 14. The Analyst's Case-List (3). *Part II.* 15. Introductory. 16. Interpretation (1). 17. Interpretation (2). 18. Interpretation (3). 19. Interpretation (4). 20. Transference and Routine (1). 21. Transference and Routine (2). 22. Transference and Routine (3). 23. Termination. 24. Psychotic Cases. 25. Relation of Theory to Practice. 26. Summary of Report. *Part III.* 27. The Therapeutic Effect of Inexact Interpretation: A Contribution to the Theory of Suggestion. 28. On the Theory of the Therapeutic Results of Psycho-Analysis. 29. Therapeutic Criteria of Psycho-Analysis. Index.

The technique of psycho-analysis is notoriously a difficult subject to condense into one book and consequently the study of the subject necessitates delving into innumerable articles scattered throughout the literature. Dr. Glover in 1928 wrote a shorter treatise on the technique of psycho-analysis, and the first part of the present volume—an expanded and revised edition of this earlier work—covers a great deal of ground in a relatively detailed way. It

reflects the change in emphasis which has taken place, from libido analysis to ego analysis and the analysis of the defences, and the greater recognition which to-day is given to the importance of counter-transference. The second half of this section, 'The Analyst's Caselist,' is an attempt to divide cases which are accepted for analysis into 3 classes—accessible, moderately accessible, and intractable. He discusses ways in which analytic technique may be modified in dealing with these respective types.

Part 2 consists of a research originally published in 1940, based on a questionnaire to a considerable number of analysts, regarding certain practical points of technique, and how they dealt with them. A summary of the replies is given and discussed. In dealing with these continually recurring problems, which are as much the subject of discussion today as then, it is evident that there was considerable difference of opinion, and that there was no rigid set of rules to which everything must be made to fit, but much variation in meeting the individual requirements of patients.

Part 3 consists of 3 papers previously published in the International Journal of Psycho-analysis, in which the nature of the therapeutic effect of psycho-analysis is examined.

This book was written chiefly for analysts, and particularly candidates commencing their clinical work. The author uses technical phraseology, and in order to follow him a considerable detailed knowledge of psycho-analytic ideas and methods is required. Even then, though the style is clear and thoughts very happily expressed, the need to conserve space leads to a paucity of illustrative case-material and lack of explanation, which at times makes his meaning obscure—a practical difficulty on which Dr. Glover comments in his preface. However, in spite of the fact that he has had to condense so much into one volume, a large amount of valuble information has been presented and many important points discussed in a stimulating way. His book will form an important addition to any analytic library.

W.A.S.

PERIPHERAL NERVE INJURIES

Peripheral Nerve Injuries. By The Nerve Injuries Committee of the Medical Research Council. Pp. 451+xvi. With illustrations £2 15s. London: Her Majesty's Stationery Office. 1954.

Contents: Part I. Methods of Investigating Nerve Injuries. 2. Rates of Regeneration. 3. Vasometor and Nutritional Disturbances. Following Injuries to Peripheral Nerves. *Part* 2. 4. Lesions in Continuity. 5. Nerve Injuries and Fractures. *Part* 3. 6. Histopathology of Nerve Injury. 7. Histopathology of Nerve Grafts as Determined by Animal Experiment. *Part* 4. 8. Causalgia—A Review of 48 Cases *Part* 5. 9. Neurovascular Lesions. *Part* 6. 10. Electrical Diagnosis of Peripheral Nerve Injury. 11. Electromyography. *Part* 7. 12. Factors Influencing Functional Recovery. *Part* 8. Results of Nerve Suture. *Part* 9. Nerve Grafting and Other Unusual Forms of Nerve Repair. *Part* 10. Open Wounds of the Brachial Plexus. Acknowledgements. Bibliography. Index. Bibliographical Index.

This most admirable book is the outcome of work over a number of years at five Peripheral Nerve Centres in Britain during and after the last war. It is not often that one can say that there is no section of a book that is not up to the highest standards demanded by medical investigations, but the authors here, under the guidance of Prof. H. J. Seddon, have produced such a volume. The work described varies from a review of methods of investigation to histopathology and the long-term results of operative treatment. Amongst the most interesting points brought out in the clinical assessment of nerve injuries is the unreliablity of the older methods of electrical testing. Electromyography has clearly proved itself to be a most useful method of investigating nerve recovery, and the earliest signs of motor recovery after nerve injury are obtained by electromygraphy, but Dr. A. E. Ritchie suggests, on the basis of thorough and painstaking work, that the usual galvanic-faradic apparatus used in neuro-muscular disorders should be replaced by a simple stimulator delivering two different lengths of shock, of approximately 1 and 300 m.sec respectively. Those who have at times been misled by the older methods will welcome this suggestion.

Professor Seddon's opening chapter gives a useful summary of anomalous muscle actions and anomalous nerve supplies, and from the clinician's point of view Dr. Barnes' chapter on Causalgia is of outstanding interest, although one must be disappointed that the treatment of this condition remains so problematical.

It is perhaps a little disappointing that in this excellent book on Nerve Injuries there is no chapter devoted entirely to the assessment of individual muscle action, so that the volume must be read in conjunction with the M.R.C. War Memorandum No. 7, which was of such great use to the profession during the last war.

The present work is not light reading by any means, nor is it intended as such, but it is the most valuable source book on peripheral nerve injuries that has been written, and contains many excellent illustrations and a very full bibliography. No one interested in a clear, scientific approach to nerve injuries can afford to miss this book.

J. Mac W. Mac G.

CARDIOGRAPHY

Cardiography. By William Evans, M.D., D.Sc., F.R.C.P. Second Edition. Pp. 212+xiii, with 378 figures. London: Butterworth & Co. (Publishers) Limited. South African Office: Butterworth & Co. (Africa) Limited, Durban. 1954.

Contents: Part I. Electrocardiography. 1. The Cardiographic Leads. 2. Deviation of the Normal Electrical Axis. 3. Ventricular and Auricular Preponderance. 4. The Physiological Electrocardiogram. 5. Altered Rhythm. 6. Congenital Heart Disease. 7. Pericardial Disease. 8. Mitral Valvular Disease. 9. Aortic Valvular Disease. 9. Aortic Valvular Disease. 10. Cardiac Infarction. 11. Hypertension. 12. Pulmonary Heart Disease. 13. The Electrocardiogram in Endocrine Disease. 14. The Electrocardiogram in Certain Other Conditions. 15. Test Electrocardiograms. *Part II. Phonocardiography.* 16. Heart Sounds. 17. Heart Murmurs. Index.

This very useful book is unusual in that it combines the study of Electrocardiography and Phonocardiography. However, the author has very definite views of his own and, although many do not agree with him, one cannot say that his views are not provocative.

His well-known insistance that the CR leads are more useful than the conventional uni-polar leads VI to V6 is certainly not accepted by most authorities working in this field.

A more useful chapter is that on IIIR (lead III with respiration) of which the author has made a special study. He also attempts to elucidate the well-known difficulty in distinguishing the S-T segment depression in ischaemia of the myocardium from other causes of S-T depression by describing the ischaemic depressions as either Sickle, Claw, Plane, or Wing depressions. However commendable this effort may be, too often, there are mixtures of Sickle & Claw or Trough & Plane or any combination, and difficulties are bound to arise.

The Phonocardiographs are good but one certainly cannot agree with the statement in the captions to the lower drawing on Page 90, that the tricuspid opening snap is responsible for the third sound in Pericarditis. It is very doubtful indeed whether the Author is justified in putting 3 after S.

The book can be recommended with confidence but the reader should rather accept the majority opinion that the V chest leads have replaced the CR leads other than in exceptional cases.

M.N.

ANTENATAL AND POSTNATAL CARE

Antenatal and Postnatal Care. By Francis J. Browne, M.D. (Aberd.), D.Sc., F.R.C.S. (Edin.), F.R.C.O.G., and J. C. McClure Browne, B.Sc., M.B., B.S., F.R.C.S. (Edin.), F.R.C.O.G. Eighth Edition. Pp. 672+viii with 94 illustrations. 37s. 6d. London: J. & A. Churchill Ltd. 1955.

Contents: 1. The History and Development of Antenatal Care. 2. Diagnosis of Early Pregnancy. 3. Examination of the Patient. 4. The Hygiene of Pregnancy. 5. The Influence of the Emotions upon Pregnancy and Parturition. 6. Maturity and Postmaturity. 7. Abnormal Presentations and Positions. 8. Multiple Pregnancy. 9. Abnormalities in the Quantity of Amniotic Fluid. 10. Haemorrhage in Early Pregnancy. 11. Haemorrhage in Early Pregnancy (continued). 12. Haemorrhage in Early Pregnancy (continued). 13. Unsuccessful Pregnancy. 14. Congenital Malformations and their Inheritance. 15. The Rhesus Factor and Erythorblastosis (Haemolytic Disease of the New-Born). 16. Haemorrhage in Late Pregnancy. 17. Haemorrhage in Late Pregnancy (continued). 18. Contracted Pelvis and Disproportion. 19. Displacements of the Uterus in Pregnancy. 20. Vomiting in Pregnancy. 21. The Toxaemias of Late Pregnancy. 22. Diseases and Disorders of the Digestive System in Pregnancy. 23. Acute Infectious Fevers in Pregnancy. 24. Diseases of the Circulatory System in Pregnancy. 25. Diseases of the Circulatory System (continued). 26. Diseases of the Nervous System in Pregnancy. 27. Diseases of the Nervous System (continued). 28. Diseases of the Nervous System (continued). 29. Diseases of the Ductless Glands in Pregnancy. 30. Diseases of the Ductless Glands in Pregnancy (continued). .31. Diseases of the Respiratory System in Pregnancy. 32. Diseases in the Urinary Tract in Pregnancy. 33. Affections of the Skin in Pregnancy. 34. Tumours complicating Pregnancy, Labour and the Puerperium. 35. Venereal Diseases in Pregnancy. 36. The Uses and Value of Radiology in Obstetrics. 37. Postnatal Care. Appendices. Index.

This classic has reached an 8th edtion and has been translated into Chinese and Spanish. The promise of immutable consistency is strengthened by the son's joining in authorship with his father. To F. J. Browne belongs the credit of raising British obstetrical writing from its rude state for, when the first edition of this work

appeared in 1935, the only scholarly works in the field were American or foreign. This edition should be studied faithfully by all undergraduates, and a reviewer reads it for his instruction. It has been revised throughout and is up-to-date.

About the time of Homer, King Solomon preached: 'Of making many books there is no end; and much study is a weariness of the flesh.' In accordance with these thoughts one feels that the most refreshing of books is the monograph which contains not so much original work as original thought. A book devoid of the last is still-born, and whoever brings together inside its cover diverse information and argument must exhibit some depth of thinking on the subject. It is fashionable for professional writers merely to add recently published matter to their previous editions.

Browne, however, is a thinker in addition to being a scholar, and his book is a precious possession. He has been regarded as the world's greatest authority on antenatal matters. He has made a great contribution as a pioneer of the new Obstetrics. Such is his authority that there is the occasional danger that his advice may unintentionally mislead many obstetricians; e.g. his statement about reducing the incidence of forceps delivery where cardiac disease is present. The simple-minded have also failed to detect his evasion of the important problem of uncomplicated hypertension in the toxaemia of pregnancy.

The curious thing about this brilliant book is that its title is no longer appropriate. The author does not devote over-much attention to actual practice in the prenatal field; he certainly does not know how a really good clinic should be conducted nor why the clinics of the western world are so unsuccessful. Because of an admiration for this book, and because heart disease today is the greatest killer in all but the best hands, one makes bold to suggest that the author should get an expert to write the section on heart disease. To preserve the balance the expert contribution on 'the emotions', and possibly even the one on radiology might be dispensed with.

O.S.H.

INTERNAL MEDICINE

Fundamentals of Internal Medicine. By Wallace Mason Yater. Fourth Edition. (Pp. 1276 + xxx, with illustrations.) New York: Appleton-Century-Crofts, Inc. 1954.

Contents: 1. Diseases of the Heart. 2. Diseases of the Blood Vessels. 3. Diseases of the Kidneys. 4. Diseases of the Blood and Blood-Forming Organs. 5. Diseases of the Respiratory System. 6. Diseases of the Digestive System. 7. Diseases of the Locomotor System. 8. Diseases of the Endocrine Glands. 9. Diseases of the Spleen and Reticuloendothelial. 10. Diseases of the Metabolism. 11. Diseases of Allergy. 12. Diseases due to Intoxications. 13. Diseases Due to Physical Agents. 14. Diseases Due to Vitamin Deficiency and Malnutrition. 15. The Infectious Diseases. 16. Diseases of the Nervous System. 17. Mental Diseases 18. Diseases of the Skin. 19. Diseases of the Ear. 20. Diseases of the Eye. 21. Dietetics. 22. Antibiotic Therapy Including the Sulfonamide Compounds. 23. Symptomatic and Supportive Treatment. 24. Inhalational Therapy. 25. Clinical Values and Useful Tables. 26. The Physician Himself. Index.

This text-book of medicine aims at presenting the fundamentals of medicine in simple and concise yet comprehensive form, and in the main it has succeeded. It certainly is comprehensive, for it includes *inter alia* chapters on the eye, the ear, diatetics and dermatology. At times it is a little too concise, for there may be only a list of names of different diseases with just enough descriptive literature to make one recognize the disease. Other parts of the book get rather more than their share. For example, there are 54 pages dealing with electrocardiography compared with 64 pages on the remainder of the diseases of the heart. But on the whole the material is fairly chosen, adequately described and accurately set out. Treatment is well discussed. The practice of dealing with the treatment of a lot of similar diseases at the end of a chapter has much to commend it and might be followed by other authors. There are many tables of differential diagnosis which are bound to be popular with the student, more especially those with their eyes on forthcoming examinations.

There is much to criticize and much to praise. Proprietary names of drugs are commonly mentioned without their official equivalent. The discussion on digitalis alkaloids is well set out but no mention is made of the different rates of excretion of the different preparations. Nor can one agree that the use of digitalis glycosides is 'more scientific' than the use of powdered leaf. The discussion on the clinical diagnosis and treatment of cardiac asthma could be improved. While the description of pernicious anaemia is good and up-to-date, sickle-cell anaemia and the other hereditary haemoglobinopathies are not nearly so well discussed. The prognosis of polycythaemia vera is unnecessarily gloomy and most workers have long since abandoned phenylhydrazine therapy for this disease as they have also abandoned the extract of yellow bone-marrow for agranulocytosis. Haemophilia is no longer believed to be due to an increased resistance of the blood platelets to disintegration. An initial dose of 6 g. of sulphadiazine in lobar pneumonia is unnecessarily heroic therapy. The statement that flat or inverted T waves in lead 1 or 11 of the electrocardiogram are frequently found in persons with neurocirculatory asthenia cannot be allowed to pass without comment. Nor should it be said that 'systolic murmurs without other evidence of heart disease do not allow the diagnosis of heart disease to be made', even though 'they are looked upon with suspicion'. The nitroglycerin test for migrane could hardly be described as useful in atypical cases. Will not 0·6 mg. of nitroglycerin produce headache in many non-migrainous subjects? While the newer drugs used in the treatment of epilepsy are well described one misses a mention of paraldehyde in the treatment of status epilepticus. On the other hand the sections on diseases of the endocrine glands and those dealing with ACTH and cortisone are good; they are worthy of special mention.

There are many contributors to this volume. More and more the modern text-book of medicine is following this pattern. It has much to commend it for, despite the criticisms of detail in this review, this book still remains a reasonable and well set-out presentation of medicine as it is practised in the United States of America.

C.M.

CLINICAL CHEMISTRY

Clinical Chemistry in Practical Medicine. By C. P. Stewart, D.Sc. (Dunelm.), Ph.D. (Edin.) and D. M. Dunlop, B.A. (Oxon.), M.D., F.R.C.P. (Edin.), F.R.C.P. (Lond.). Fourth Edition. (Pp. 320 + vii, with figures. 21s.) Edinburgh and London: E. & S. Livingstone Limited. 1954.

Contents: 1. Introduction. 2. The Collection and Preservation of Samples. 3. Water and Electrolyte Metabolism. 4. Neutrality Regulation. 5. Carbohydrate Metabolism. 6. Tests of Renal Function. 7. The Proteins of Blood Plasma. 8. Tests of Gastric Function. 9. Tests of Pancreatic Function. 10. Tests of Hepatic Function. 11. The Cerebro-Spinal Fluid. 12. Assessment of Thyroid Function. 13. Calcium and Phosphorus. 14. Hormone Abnormalities. 15. Nutritional Deficiencies. Appendix I. Appendix II. Index.

This classical publication has been revised as compared with its 3rd edition of 1949. The excellent standard of previous editions is maintained, and the practical nature of the book for daily reference is unaltered.

For the intern it is essential. For the general practitioner it is a necessity—if only for chapter II, which gives a clear account of the proper collection and preservation of specimens. For the various specialities the 4th edition is a readable account of the recent advances as well as the established practices in Clinical Chemistry.

Three new chapters are added, viz. Assessment of Thyroid Function, Hormone Abnormalities, and Nutritional Deficiencies. The chapter on miscellaneous items has been abolished. These new chapters give a brief and modern account of their subject matter.

Minor criticism may be levelled at particular points. In chapter II there is no mention of the use of sodium fluoride tubes for the preservation of cerebrospinal fluids, on which 'sugar' determinations are often requested after a delay of 24-48 hours in postage. One would like to have seen it made clear that urea concentration or urea range tests should not be undertaken unless previous examination has established the blood-urea concentration level.

Chapter XII suggests that chemical analysis of protein-bound iodine (as opposed to radio-activity methods) are not suitable for routine use. This reads somewhat 'Irish' as a subsequent paragraph indicates. Furthermore, an accurate chemical analytical method which may be adopted for routine use in the bigger laboratories is the alkaline incinerator technique of Salter and McKay.

The Appendix I has been rewritten so as to be a more comprehensive account of technical methods. There is an unwise trend to describe principles of some procedures without detailing the practical techniques.

The publishers are to be commended for their fine workmanship. The book is produced with the optimal criteria for reference purposes, the size being demi-octavo, the print 11 on 12 Baskerville type and the set, to 24 pica ems.

R.S.